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TEXTBOOK OF OPHTHALMOLOGY

IN THE FORM OF CLINICAL LECTURES

BY

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

*WITH ONE HUNDRED AND EIGHTY SIX ILLUSTRATIONS IN THE TEXT
AND THIRTEEN COLORED PLATES*

REVISED EDITION

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TO

PROFESSOR PAUL EHRLICH

THE GENIUS OF INVESTIGATORS AND THE LEADER
IN MANY FIELDS OF MEDICAL SCIENCE
AS A TOKEN OF
HIGH ESTEEM, INEXTINGUISHABLE GRATITUDE
AND TRUE FRIENDSHIP
THIS WORK IS DEDICATED BY
THE AUTHOR

PREFACE

This Textbook of Ophthalmology is intended primarily for those students whose interest and attention incited its commencement. Shortly after I undertook the clinical lectures on ophthalmology at Greifswald, these students petitioned me that they might possess in permanent form the instruction I had given them, and that the latter might be kept in the style I had been accustomed to use in my lectures. I have complied with their desire, not because there is a dearth of ophthalmological textbooks, but that I might afford these grateful and diligent students of mine the possibility of taking a personal souvenir with them in their later life work. The form I have chosen seems suited for this purpose in spite of manifest faults. When one or another of them in the future may look over the clinical cases here recorded, when in his mental vision he views the life in the clinic, when the personality of his teacher is with him again, many a recollection from bygone days will be quickened, for the most delightful period in the life of a physician is the time when, still young and with perfect ideal, he visits the clinics and treats his first patients without responsibility.

Didactically, also, the form of the clinical lecture seems to me to be proper for a textbook of ophthalmology, even though the details of clinical histories must be omitted because of limited space. With changes in the small organ so easily overlooked, it is important that those who wish to learn, or to freshen and enlarge their knowledge, should always, in each type of disease, refer systematically to all that may be considered in such cases. The form employed certainly involves the danger of a certain monotony for the reader, but it is hoped that this will be somewhat ameliorated by the direct, living intercourse between him and the book. At any rate it is always easier to remember the things to be observed in one's own practice from the individual description of certain cases than from a summary discussion of a certain subject. Hence I hope that not only the student, but the physician as well will look with a kindly eye upon the pedagogy in this book. For it may say of itself that it is born of a glowing love of science, and is animated only by the wish to be of service to the physician and the patient, to the learner and the sufferer.

But the book desires still more. It desires to be welcome to the younger men of our specialty who themselves teach ophthalmology. I agree with my colleagues in believing that the clinical instruction in ophthalmology should be extended over at least two semesters, an advancement that is justified by the growth of our field. If the physician is to obtain an adequate insight into our specialty it takes time. Thus it has been my endeavor throughout to hold in form and content as rounded a clinic as possible.

The arrangement of the material chosen has the advantage that the teacher who takes a pathological case for the text of a clinical lecture will always know exactly what he has to teach without delay or hurry. It is easily possible for the master of his specialty to devote sufficient attention to the variegated changes of the pathological picture and still have time enough for the rarer diseases, or to add, or interpolate minor cases for the purpose of repetition, or demonstration.

As I consider that the teaching of ophthalmology should not be purely theoretical, but that the student should enjoy opportunities for practical treatment and be able to see many operations, I have endeavored to present the subject in as popular language as possible. This may be a weakness in the book, but, as it is to be a textbook from which the beginner is to learn, I willingly accept it, for I, like other teachers, take pleasure when my students learn easily. As the instruction is to be as didactic and vivid as possible, a textbook must be written fluently and be easy reading; clearness and simplicity are the fundamentals of clinical pedagogy.

As an ophthalmologist I consider it self evident that the book should not be printed on any paper with a glaze that renders prolonged reading painful in any light. As ophthalmologists we should be promoters of the hygiene of the eye before all else.

I have restricted the number of illustrations. There is very much that cannot be adequately learned of diseases of the eye from a picture.

One feeling has never left me. From whatever point of view a textbook may be written, it must ever remain imperfect and not solely because of the necessary limitations of space. The incompleteness of our knowledge is a sorrow to the scholar, the inadequacy of our therapeusis bears sorely on the mind of the physician, and only the pleasure of working with and serving youth rich in future promise encourages the teacher in issuing this book.

PAUL ROEMER.

TRANSLATOR'S PREFACE

At the time of his lamented death, the late Dr. P. W. Shedd was engaged in the translation of this work and had completed the first ten lectures, about seventy pages. These are much as he left them, except for a slight recasting, which seemed necessary to preserve uniformity of style throughout.

This is the first attempt, of which I am aware, to substitute concrete instruction for abstract discussion in a textbook of ophthalmology. In all branches of medicine oral instruction has changed during the last generation from the delivery of didactic lectures to the gathering of students about the bedside of patients for clinical demonstration, but the textbooks the same students study still maintain the form of more or less terse, compact statements of abstract fact, and rely on grace of diction to remove the dryness of the exposition. Roemer has made the departure of presenting the same ophthalmological facts in the concrete form employed in the clinical lecture, and has done this so successfully that it frequently seems to the reader that the lectures must have been actually delivered and reported by a stenographer; the frequent repetitions and side remarks, so often used unconsciously by clinical teachers, occur so naturally that it is hard to believe they are all intentional. It is unnecessary to dwell upon the advantage to the student of a definite clinical picture of a disease firmly impressed upon the mind, this is generally acknowledged. Such a picture Roemer tries to impress, and then he points out the variations that are to be met with. It has not been an easy task to reproduce the power, vividness and force of the original German, in which the pen pictures are so lifelike that they seem to bring the individual patients, who form the texts of the clinical lectures, distinctly before the reader. How well, or ill, I have succeeded I shall presently learn from the reviewer.

The specific treatment of disease in accordance with its etiology is a subject in which the author is an enthusiast, and he imbues even the skeptical reader with some of his enthusiasm. He has one fault in common with many writers, a certain provincialism that leads him to undervalue some procedures that did not originate in his native country and to omit all mention of others. In a few instances I have added footnotes to supply some of these omissions.

In view of the fact that medical orthography is in a rather chaotic condition at the present time, and as the dictionaries usually accepted as authorities vary enormously among themselves in this respect, I may state that I have tried to conform in my spelling of medical terms to the recommendations of the Committee on Nomenclature of the American Medical Association, published in the Transactions of the American Ophthalmological Society for 1909, as this is the most authoritative pronouncement in regard to the subject which has come to my knowledge. Wherever variations from these recommendations may appear in the text, they have not been made intentionally.

MATTHIAS LANCKTON FOSTER.

211 CENTRE AVENUE, NEW ROCHELLE, N. Y.

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I

Introduction to the Methods of Examining the Anterior Segment of the Eve

1. Anatomy and Clinical Examination of the Normal Conjunctiva, the Cardinal Symptoms of Conjunctivitis and the Inquiries to be Made on Examining an Inflammation of the Conjunctiva

We may very properly begin with the study of the morbid affections of the conjunctiva and cornea, for of all the abnormal changes that trouble the eye, the external diseases, those of the conjunctiva and cornea, are the commonest.

Diseases of the conjunctiva include about 30%, those of the cornea about 20%, of all forms of ophthalmologic troubles. These external diseases are of import to the general practitioner, since precisely these morbid conditions are suited to his therapeutic care and the patients so afflicted will demand his treatment.

Of the diseases of the conjunctiva inflammations are most frequently encountered, and therefore demand our attention first. Before commencing the study of these conjunctival inflammations, it is imperative that the student acquire

1. An acquaintance with the normal anatomy of the conjunctiva, as fundamental to further study of the subject-matter.
2. A knowledge of how to investigate clinically the normal conjunctiva in the living subject.
3. An ability to recognize an inflammation of the conjunctiva as compared with the normal appearance of this membrane, i.e., an ability to recognize the cardinal phenomena met with in almost every inflammation of the conjunctiva.
4. A knowledge of the order in which to interrogate the patient in each examination of a case of conjunctival inflammation. This must be learned if we desire to establish an accurate diagnosis.

We shall try to meet these four requirements now, in the beginning of our course, and turn first to the normal anatomy of the conjunctiva.

I. The Normal Anatomy of the Conjunctiva

The conjunctiva is in the form of a sac, or bag, the conjunctival sac, split by the palpebral fissure, which covers the surface of the eye, the posterior surface of the eyelid, and the contact points of union between the two. Hence we may differentiate it into 1, a conjunctiva bulbi; 2, a conjunctiva palpebrarum; 3, a conjunctiva fornicis, the transition fold.

The conjunctiva bulbi has a surface of stratified flat epithelium, with occasional, isolated, simple, tubular glands (*Manz's* glands). No goblet cells are present, and because of this the conjunctiva scleræ does not appear to be as moist as the conjunctiva tarsi and the transition folds. In so far as the conjunctiva bulbi covers the sclera, we call it the conjunctiva scleræ. It should not be thought, however, and this is a matter that demands immediate emphasis, that it possesses an aperture corresponding to the cornea. The conjunctiva scleræ extends, rather, in continuity over the cornea. The epithelium of the cornea and its superficial strata belong embryologically to the conjunctiva. The significance of this fact will be understood presently.

It can be seen from histologic preparations that the epithelial surface of the conjunctiva tarsi is composed of polystratified cylindric epithelia and contains a greater or less number of goblet cells. The conjunctiva tarsi is firmly attached to its subjacent tissue, and pathologic changes only are capable of loosening it somewhat. In the posterior part of the tarsal conjunctiva we find the small, ramifying, tubular glands of *Krause* situated above the *Meibomian* glands.

Another feature that is to be noted is, that, in a more or less extensive portion of the conjunctiva tarsi, minute, but genuine, papillæ are to be found, slight elevations with vessels and surrounding stroma, between which the epithelium dips somewhat downward. Under normal conditions the mucous membrane remains perfectly smooth, but in inflammation these papillæ enlarge, and the mucosa then presents, as will be seen later, a velvety appearance, or larger prominences develop, as in vernal catarrh, or in trachoma.

Under normal conditions, there is at the transition folds an abundance of conjunctiva which possesses numerous elastic fibres and is also covered with a stratified, cylindric epithelium. Moreover, it will be observed microscopically that beneath the epithelium is a layer of adenoid tissue that contains lymphatic cells and also, in most adults, isolated and very minute follicles. At the transition folds papillæ are lacking and only small plicæ are to be seen.

The vascular supply of any mucous membrane is of great importance, both to the nutrition and to our comprehension of the morbid conditions that affect it. The essentials of this can be seen at once in Fig. 1. On the upper lid, bloodvessels penetrate the tarsus from without inward, 2 to 3 mm. behind the palpebral margin, along a furrow which will presently be demonstrated on the living subject. These vessels belong to the arcus tarseus inferior, and the shallow furrow that runs parallel to the margin of the lid is called the sulcus subtarsalis. The bloodvessels of the arcus tarseus superior pass in an analogous manner to the conjunctiva at the upper margin of the tarsus. In the lower lid we see only a single vascular arch along the convex margin of the tarsus. The transition folds of the upper and lower lids are supplied by these vessels. The conjunctiva bulbi derives its blood-supply chiefly from the vessels of the transition folds. These form the posterior conjunctival vessels. There is still another vascular system, that of the anterior ciliary vessels, which pass from the four rectus muscles beneath the conjunctiva, through which they are visible as shimmering, bluish lines. These anterior ciliary vessels disappear in the vicinity of the margin of the cornea, where they perforate the sclera and continue into the eye to supply the iris and the ciliary body with blood. Before they pass through the sclera, these anterior ciliary vessels send branches backward in a course opposite to that of the other conjunctival vessels, and are termed the anterior conjunctival vessels. All of these end at the edge of the cornea in capillary loops, which form the marginal vascular anastomosis of the cornea.

II. Clinical Examination of the Normal Conjunctiva.

With the patient seated and his eyes open we see the lustre of the cornea, and, to a greater degree, the sclera; or, as the laity call it, the "white" of the eye. The sclera is covered by a delicate, translucent membrane, the conjunctiva scleræ.

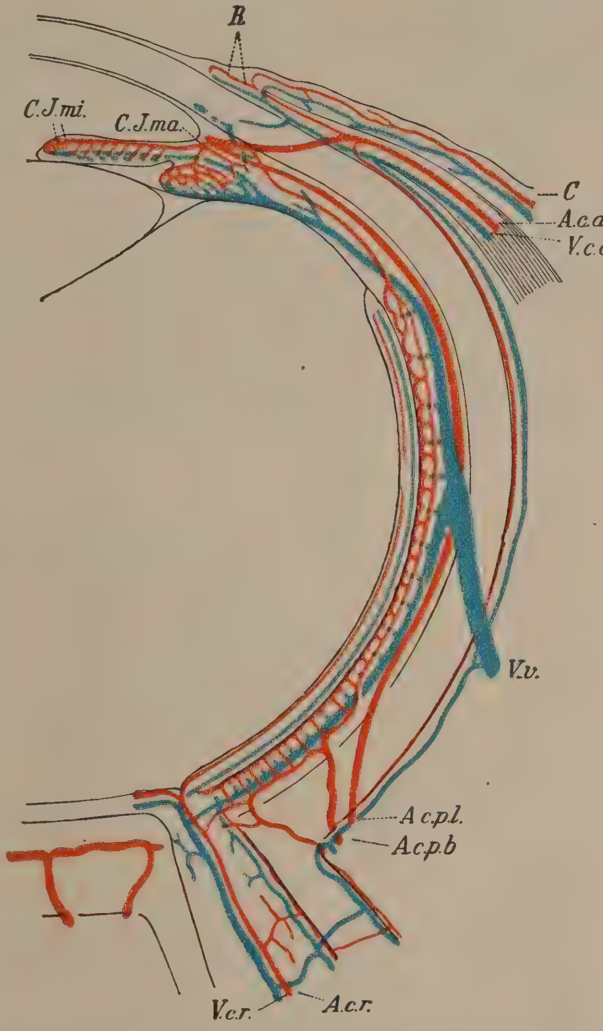

FIG. 1.—Schematic Section Showing the Distribution of Vessels According to *Leber*.

A.c.a.: Arteria ciliaris antica. *A.c.p.b.:* Arteria ciliaris postica brevis. *A.c.p.l.:* Arteria ciliaris postica longa. *A.c.r.:* Arteria centralis retinæ. *C.:* Conjunctival vessels. *C.J.ma.:* Circulus arteriosus iridis major. *C.J.mi.:* Circulus arteriosus minor. *R.:* Capillary anastomosis between the conjunctival and the ciliary vessels. *V.c.a.:* Vena ciliaris antica. *V.c.r.:* Vena centralis retinæ. *V.v.:* Vena vorticosa.

The conjunctiva scleræ—and this deserves note—is attached to the sclera by a very loose connective tissue. By pressure with the finger against the edge of the patient's lower lid, the conjunctiva scleræ may be moved to and fro, and when the eye is cocainized the conjunctiva bulbi can be raised with the forceps into folds.

This loose attachment of the conjunctiva to the sclera is to secure the freest movement to the eyeball.

This loose connection with the sclera is advantageous to us in that by drawing together neighboring parts of the conjunctiva we are able to perform plastic operations to repair losses of substance.

In the vicinity of the cornea, the attachment of the conjunctiva scleræ becomes firmer, and at the corneal margin the epithelium of the conjunctiva passes smoothly over into that of the cornea. At the margin of the cornea the conjunctiva is particularly tenuous and permits the sclera to gleam through distinctly. Altogether, we find the conjunctiva to be a thin, translucent membrane, in which are individual bright red bloodvessels. In elderly people it may have a dulled appearance in just one characteristic place, as I will demonstrate in the patient here before us.

A small part of the conjunctiva scleræ is conspicuous, because of its yellow tint in contrast with its surroundings in the portion corresponding to the palpebral fissure, at both the outer and the inner edge of the cornea. This area is rendered prominent in the otherwise quite normal conjunctiva by its cloudiness and yellowish-gray thickening. This peculiar formation is apt to become still more apparent when the conjunctiva in its vicinity is reddened and inflamed. The persons affected occasionally seek professional advice on account of this petty cosmetic disfigurement. The area thus altered is of the form of a triangle with its base toward the cornea, its apex toward the canthus. This is called a pinguecula and is a sort of callous, or hyaline thickening of the conjunctiva, due to atmospheric influences—wind, smoke, and dust. It is usually met with in elderly persons who live much out of doors.

If the conjunctiva scleræ is now traced, not in the direction of the cornea, but toward the periphery, it will be seen that in the inner canthus the conjunctiva bulbi forms a small, vertical fold lying on the eyeball, which is called the plica semilunaris. This is a shrunken remnant of the palpebra tertia, the nictitating membrane of the lower animals.

Still farther inward from the plica semilunaris is a small reddish elevation, the caruncle, which occupies the horseshoe-shaped notch at the inner canthus. The caruncle is merely an islet of skin, provided with sebaceous glands and fine hairs. Of the entire conjunctiva of the eye, we ordinarily see only a small part of the conjunctiva scleræ. If we wish to look at the other parts we must evert the lids, a procedure I shall demonstrate immediately, for it is often incorrectly performed.

I place my forefinger or thumb on the middle of the lower lid, so that its tip touches the palpebral margin. Then I ask the patient to look up, and at the same time draw the lid gently downward and somewhat away from the eye.

Frequently the finger is placed too low down, even on the cheek, and then nothing of the conjunctiva can be seen, no matter how nearly vertical is the patient's line of vision, and the net result is the annoyance of the patient from the traction on his face. If, however, the eversion of the lower lid has been correctly performed, first the lower portion of the conjunctiva scleræ, then the conjunctiva of the transition fold, and the conjunctiva palpebralis become visible. Shimmering through the lower transition fold may be seen the dense venous plexus that lies beneath it, and the fascia.

The most notable characteristic of the transition fold is its roominess and the mobility of its conjunctiva, which, with the loose attachment of the conjunctiva to the sclera, assures free movement to the eye.

In this way the eye is enabled to move independently of the lids, the folds of the transitional parts smoothing out or folding together, as occasion may require.

The case with the conjunctiva tarsi is quite different; this is not movable, but, on the contrary, firmly adherent to the subjacent tarsus, so we are unable to slide neighboring parts to cover defects, as in the conjunctiva bulbi. The conjunctiva tarsi is smooth and through it shimmer, like a row of parallel bright lines, the *Meibomian* glands which lie in the tarsus. Now the rest of the conjunctiva remains to be examined.

To bring the upper part of the conjunctiva scleræ into view, I place my thumb quite gently upon the upper lid, tell the patient to look directly downward, and at the same time lift the lid. If we wish to see the conjunctiva of the upper lid and transition fold, the upper lid must be everted. This is more difficult than on the lower lid.

Again I direct the patient to look down. I seize the lashes in the middle of the margin of the upper lid with the thumb and forefinger of one hand and draw the lid somewhat downward, and at the same time somewhat out from the

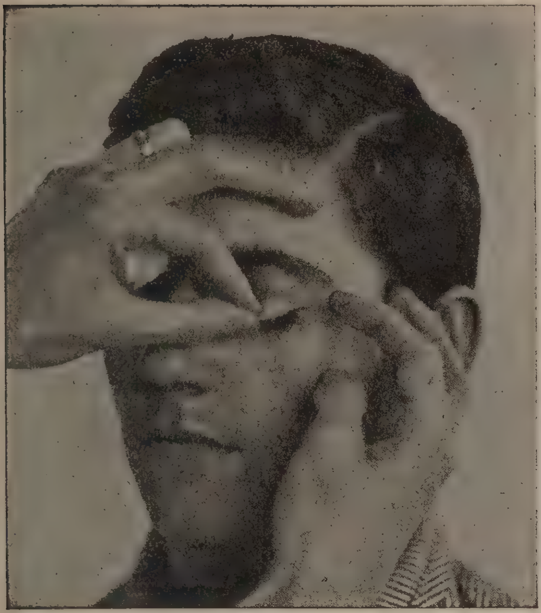

FIG. 2.

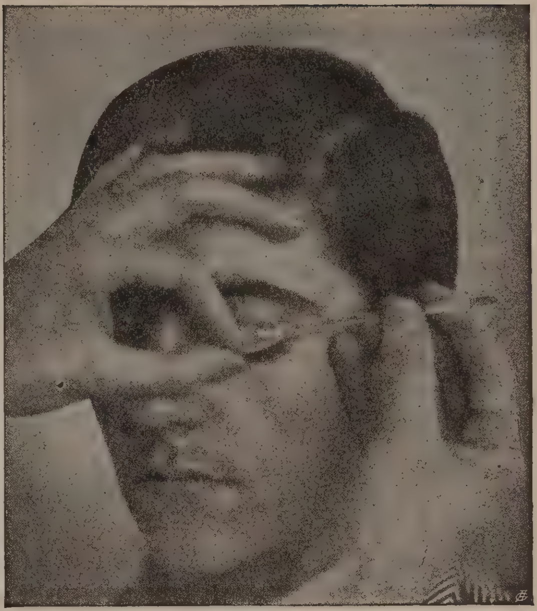

FIG. 3.

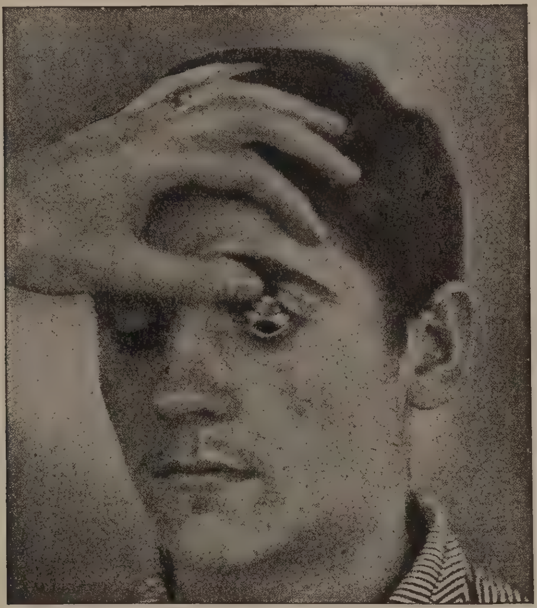

FIG. 4.

Eversion of the Upper Lid.

eye. I then place the tip of the index finger of the other hand on the middle of the drawn-down lid, i.e., on the upper edge of the tarsus, press the tip of the finger somewhat backward, and turn over the lid about this as a fulcrum. In this way the conjunctiva tarsi of the upper lid is brought into view. Under normal conditions the *Meibomian* glands may be seen shimmering through it. Another point of interest is the sulcus subtarsalis, already mentioned, about 3 mm. behind the palpebral margin, where the branches of the arcus tarseus inferior pass through the tarsus. This is the favorite place of lodgment of small foreign bodies. When any one walking in the street has a bit of dust, a cinder, or a particle of stone fly into the eye and lodge beneath the upper lid, the sulcus subtarsalis must be remembered.

The attempt to evert the lid should not be too prolonged, for, aside from the discomfort it causes the patient, a prolonged retention of the lid in an everted condition readily leads the mucous membrane to become increasingly red, a condition we cannot call inflammatory. Inversely, if too great pressure is made on the lid, the mucous membrane becomes bloodless and whitish, which may lead the tyro to diagnosticate the presence of cicatrices. It is to be recommended that during the investigation the pressure of the finger be somewhat lessened, and that the position of the fingers be changed. If, now, the patient looks strongly downward, and the everted lid is pressed firmly against the margin of the orbit, the upper transitional fold comes into view. In some cases it is necessary to doubly evert the upper lid in order to obtain a more accurate examination of the transition fold.

III. The Cardinal Signs in Conjunctival Inflammation

The normal condition and appearance of the conjunctiva may now be compared with that of a rather extensive and diffuse inflammation of the conjunctiva in a patient. Although the student knows nothing as yet of the disease of the conjunctiva, for he comes to the clinic not because he knows, but to learn, still he will be able at once to recognize the three ever-recurrent cardinal signs of a distinct and extensive inflammation of this membrane, viz.: redness, swelling, and abnormal secretion.

The conjunctiva of this patient is very red in comparison with the normal mucous membrane, because of the increased and long-continued hyperæmia due to the inflammation. It is also swollen. In grave inflammations the conjunctiva bulbi may even be raised from its bed by exudation. If, as in the present case, the swelling is confined to the conjunctiva tarsi, we recognize it as such because we can no longer see the *Meibomian* glands distinctly through the thickened turbid membrane, or perhaps may not be able to see them at all. Furthermore, we find in this patient an increased secretion from the mucous membrane which is not present in the healthy eye. This secretion gathers chiefly in the canthi.

IV. The Sequence of Interrogations in Examining Each Case of Conjunctival Inflammation

Having thus established the presence of these three cardinal signs, we know that this patient has an inflammation of the conjunctiva, but we do not know much about the morbid condition in its entirety. We know neither how long it has existed, nor the form of inflammation present, nor the etiology of the disease. In making inquiries, it is advisable to follow a definite series of questions in the following sequence:

1. Acute or Chronic Inflammation?

It is best to learn first whether the inflammation has been of short or long duration; whether it is acute or chronic. As soon as we know this we are able to exclude in any given case a large group of inflammations, and this is of importance in differential diagnosis, as we shall see later. To learn whether the inflammation is acute or chronic, it is necessary only to ask the patient.

There are objective changes in the mucous membrane by means of which we may recognize chronic forms of inflammation, but we shall omit this individualizing study; it is of more importance just now to become accustomed to our plan of inquiry.

2. Catarrhal, Croupous, or Purulent Inflammation?

Having learned by inquiry of the patient whether this is an acute or a chronic inflammation, the thoughtful physician will question involuntarily concerning its form. The pathologist recognizes three forms of inflammation of the mucous membranes—the catarrhal, the croupous, and the purulent.

In the catarrhal form, the exudate consists essentially of fluid, and usually the surface epithelium is covered with mucus in larger quantity than normal.

If there is an excretion of fibrin in the fluid exudate, so that it coagulates, serofibrinous or fibrinous exudates are formed which we designate as croupous. In such cases whitish flakes will be seen lying on the mucous membrane, or, if the inflammation is more intense, membranes will be formed that are firmly attached.

If leucocytes are found in great numbers in the fluid exudate on the surface of the mucous membrane, we call it pus, and speak of a purulent inflammation.

This clinico-anatomical diagnosis of a conjunctivitis is the question most easily answered in our systematic diagnosis. In our patient there is no membrane on the conjunctiva, nor anything much resembling pus; the secretion is rather of a mucous or serous character and therefore the case is to be diagnosed as one of catarrhal conjunctivitis.

Beautiful as these definitions are, easy as it seems from a purely clinical standpoint to make such a diagnosis of catarrhal, croupous, or purulent conjunctivitis, yet there are many transition forms in this nosologic group which frequently cannot be sharply differentiated. This is due in part to the stage in which we meet with the case of conjunctivitis. Take a case of simple cold in the head. At first the secretion from the nasal mucous membrane is watery, later it assumes, perhaps, a purulent character. Shall we call this inflammation of a mucous membrane purely catarrhal? The conditions are quite similar in a number of forms of conjunctivitis.

The complete clinico-anatomic picture of a conjunctivitis is, however, dependent on another factor, for a biologic law takes part in these inflammations of the conjunctiva, the significance of which demands attention if we are to understand ophthalmic pathology and keep pace with the progress of ophthalmic therapy. Every conjunctivitis is simply the expression of a reaction between the organism and the mucous membrane on the one hand, and the morbific factor that develops inflammation on the other. Preeminent among the latter are microorganisms, and the inflammatory reaction produced depends on the general and local susceptibility of the individual, as well as on the virulence of the morbific agent. Thus the diphtheria bacillus may excite in one person a grave croupous conjunctivitis that endangers the eye, while in another it causes a mild attack of disease and, after the patient's recovery, may remain in the eye for weeks without producing any reaction. Likewise, the conjunctiva of many persons is immune to pneumococcic infection, while the same germs, transferred to other persons, excite a violent conjunctivitis. Hence, a clinically mild catarrhal conjunctivitis may be due to an infection that has no serious consequences to the patient himself, but may endanger those about him. I myself know of a family tragedy in which a mild conjunctivitis in one of the children, not recognized as diphtheritic, gave rise in the other children to diphtheria of the throat, from which they died.

Having acquired this information—knowing that the etiologic agent may develop in the conjunctiva in one case a mild catarrhal syndrome, in another a grave croupous inflammation—it becomes self-evident that the answer to our second question, whether the inflammation is catarrhal, croupous, or purulent, does not suffice. It is of even greater importance to know the true cause of the inflammation, for the final and weightiest component of our diagnosis is the determination of the etiology of the conjunctivitis in question. With this we arrive at our third and final query—the etiology of conjunctival inflammations.

3. *What Causes the Conjunctival Inflammation?*

An inflammation of the mucous membrane of the eye may arise from two different causes, which may combine in the later stages in a variety of ways.

A conjunctivitis may be caused either by purely physico-chemical irritants, or by an infection. It must be understood that the chemical, toxic effects of the bacteria and of their metabolic products are present in bacterial infections. Furthermore, it must be borne in mind that a mucous membrane injured by chemical or physical means invites bacterial invasion, so that the two factors may form the most varying alliances. But it is better just now, for practical reasons, to consider these two causative agents separately, because we then obtain a viewpoint at once from which to select the line of our investigation of the etiology of a conjunctivitis.

Our final question is, therefore, whether the inflammation of the conjunctiva is due to purely physico-chemical causes, or to infection. The fact is, that quite a number of acute and chronic inflammations of the conjunctiva are caused by physico-chemical irritants. Thus, foreign bodies, especially beards of grain, when they remain for a considerable time in the transition fold, may excite severe inflammations of the eye and are to be thought of in cases of violent conjunctivitis of only one eye.

Exposure to dust-laden air is capable of exciting a conjunctivitis, or of aggravating an inflammation already present. Eyes reddened by nocturnal celebrations are not altogether unknown among students. I have been able to demonstrate experimentally that even sterilized dust augments the bacterial content of the conjunctival sac by its irritant effect.

Another form of conjunctivitis may be caused by caterpillar hairs. The most violent inflammation of the eye may be excited by dust mixed with such hairs in woods where the procession caterpillars congregate. In the Orient the dust blown about not rarely contains large quantities of extremely minute cactus thorns. In hay fever, the acute conjunctivitis is caused by particles of pollen from the grasses, and the pollen from other flowers may have a like irritant effect.

It is self-evident that all chemically irritating substances may excite an inflammation of the conjunctiva. In practice, we meet with such causes as the dust from lime, cement, artificial fertilizers and snuff. Malingerers and hysterical persons not infrequently place substances of this sort in the conjunctival sac intentionally. In accident cases, a conjunctivitis of one eye confined to the lower part of the conjunctiva excites our suspicion. There are also drugs which may be responsible for a conjunctivitis. We must not bring any ointment containing chrysarobin into contact with the lids, because it may cause an intense inflammation of the conjunctiva which can extend deep into the eye. Even so necessary a remedy as atropin excites a conjunctivitis in not a few patients, associated, usually, though not always, with development of follicles. Furthermore, no calomel should be dusted into the conjunctival sac of a person who is receiving internally iodide of potassium, as mercuric iodide may be produced which will occasion a violent conjunctivitis.

In many occupations forms of conjunctivitis are met with that are traceable to radiant heat, or to chemically active, ultraviolet rays of light. In this category, glacier catarrh and snow blindness are included.

Finally, here belong, in part, those inflammations of the conjunctiva which are caused and maintained by the scraping of abnormally placed cilia, imperfect closure of the eyelids, and anomalies of position and occlusion of the puncta lacrymalia. The group of inflammations of the conjunctiva that may be due to physico-chemical agents is not small, and it is important to bear this etiologic factor in mind.

The percentage of cases in which the conjunctivitis is due to bacterial infection is still greater. It may be said that more than two-thirds of all inflammations of the conjunctiva are caused by bacteria. The history helps us to determine whether a conjunctivitis is due to physico-chemical irritation, or to an infection, but the real cause can be determined only by a bacteriological examination of the secretion and of the surface of the conjunctiva, by which we learn whether bacteria that excite inflammation are present or not. The methods employed in doing this and the points of view to be considered, will be indicated in the lecture when the individual study of conjunctival diseases will be commenced. I will repeat the questions to be asked when we meet with a conjunctivitis: Is the inflammation acute or chronic? Is it catarrhal, croupous, or purulent? And, finally, Is its cause a physico-chemical irritation, or is it probably an infection? These are questions to be always borne in mind.

2. Clinical Examination of the Normal Cornea and its Physiologic Clouding in Old Age

The anatomical and embryological fact has been briefly mentioned that the conjunctiva scleræ extends over the surface of the cornea and forms an integral part of the cornea itself. This conjunctival portion of the cornea consists of the epithelium and *Bowman's* membrane, to which are to be added the most anterior lamellæ of the cornea in its marginal portion. This fact is explanatory of a clinical phenomenon essential to the understanding of conjunctival diseases, indeed of all external affections of the eye, and we will continually meet with it from now on.

A large number of conjunctival diseases tend to invade the cornea, and this involvement of the cornea appears in clinical syndromes of the greatest variety. It is therefore self-evident that when we wish to pass from the normal conjunctiva to the study of its diseases, we must not confine ourselves to the clinical examination of the conjunctiva of the lids, the transition folds and the sclera, but must also learn to investigate the normal cornea.

The cornea is set like a watch-glass in the sclera, the anterior layers of which overlap its margin. The clinical importance of this anatomical fact will be touched upon at various times in the description of operations. The transition of the fibres of the sclera into those of the cornea is very gradual.

The layers of the cornea, knowledge of which is essential, are from before backward:

1. The epithelium. This is a stratified pavement epithelium. Its deepest

cells are columnar, then come cells that are more spherical, and above these they are flat, so that the cornea has a very smooth surface.

2. *Bowman's* membrane. This, a structureless membrane of slight elasticity, resembles in development and nature the uppermost homogeneous layer of the parenchyma of the cornea. This explains why *Bowman's* membrane is firmly attached to the subjacent tissue and more loosely to the epithelium. In certain pathological conditions, the epithelium may be separated from it.

3. Next to *Bowman's* membrane lies the substantia propria, the parenchyma of the cornea. This, the true corneal tissue, is composed of delicate fibrillæ of connective tissue, generally arranged parallel with each other, though cross fibres are also numerous. These fine fibrils are bound together in lamellæ by a cementing substance. Between the lamellæ we meet with the corneal cells, which are also known as fixed corneal corpuscles, flat cells with large nuclei. In flat sections it can be seen that they are interjoined by numerous processes. When sections are prepared for microscopic work, these cells shrivel and leave minute spaces between the lamellæ, which once were erroneously supposed to be nutrient channels. It was thought that the cornea contained a network of nutrient spaces and canals, but this is not the case, a matter of importance to the concept of nutrition, as will presently appear.

In addition to these corpuscles, we meet with, here and there in the cornea, wandering cells with single nuclei. We shall be occupied with these cells repeatedly from now on, for it is to them that the chief part is assigned in inflammation of the cornea, when they emigrate from the marginal vessels into the parenchyma.

4. *Descemet's* membrane, a second structureless membrane, forms the posterior boundary of the parenchyma. It displays great powers of resistance. Often, when the parenchyma has been destroyed over large areas, we find *Descemet's* membrane the sole barrier.

5. Finally, *Descemet's* membrane is covered by a single layer of smooth cells with roundish nuclei.

Under normal conditions there are no bloodvessels or lymph channels in the cornea. The marginal vascular network of the conjunctiva ceases at the limbus. The cornea is richly supplied with nerves which come radiating from the ciliary nerves and form numerous anastomoses beneath *Bowman's* membrane. They also pass through the latter and ramify between it and the basal cells in a subepithelial terminal network.

The nutrition of the cornea is of much interest in the physiology of the eye and a knowledge of it is necessary to the understanding of corneal affections. A priori, the cornea may gather its nutrient material from two sources: either from the network of vessels about its margin, or from the aqueous behind it.

If we divide the vessels at the margin of the cornea in vivo, no perceptible injury results. If we protect an enucleated eye from evaporation, the cornea remains clear a long time. Hence, we may conclude that under normal conditions the nutrition of the cornea is relatively independent of the marginal network of bloodvessels.

The conditions are quite different, however, when the cornea is inflamed. In such cases we find, by anatomic investigation, that fibrin formations are to be met with in its parenchyma, and there is no doubt that the fibrogenic elements enter the cornea from the bloodvessels.

Protective substances also enter the cornea from the blood, together with the fibrin, as may be shown by the following experiment: If some diphtheria toxin is injected with a very fine needle into the cornea of two rabbits, and then one rabbit receives an intravenous injection of diphtheria antitoxin, the cornea of the latter animal will remain perfectly clear, because the antitoxin has reached the cornea and made the poison harmless, while in the other the cornea becomes very cloudy.

We occasionally see, after severe subconjunctival hemorrhages, that derivates of the coloring matter of the blood diffuse into and stain the cornea.

From these facts it may be concluded that, under certain circumstances, the marginal network of vessels takes part in the nutrition of the cornea. It is not for nothing that we frequently meet with a new formation of vessels in the cornea in many inflammations, the only reason for which can be to aid in overcoming the disease, and in the preservation of the corneal tissue when the normal nutrition no longer is sufficient. I have therefore formulated the theory that the nutrition of the cornea through the marginal network of vessels is demanded chiefly when inflammation is present, but that under normal conditions its nutrition is derived principally from the anterior chamber.

It was formerly thought that nutritive fluids circulated through canals in the cornea, but modern anatomical research has shown that such interspaces resembling canals do not exist between the cells and the ground substance of the cornea, and we are unable to perceive any movement of fluid in the tissue. But soluble substances like fluorescin, which enter the cornea, spread by diffusion uniformly in all directions. In addition to this, according to *Leber*, the intact cornea is absolutely incapable of filtration under the pressure that exists within the eye. We may therefore assume with certainty that the nutrition of the cornea is accomplished primarily by molecular forces, and that these are as efficient from the marginal network of vessels as from the anterior chamber. Two processes are involved: imbibition and diffusion.

To demonstrate the former, place some freshly prepared corneas of animals, some in water, some in isotonic, and some in hyperisotonic solutions of salt. In all these solutions the cornea becomes enormously swollen, which shows that the corneal substance has the property of eagerly saturating itself with any surrounding fluid, to imbibe water. At the same time the cornea becomes cloudy. In a normal condition, it will take up 80% of water, of distilled water 96%, its thickness increasing eightfold. As this swelling is not dependent on the osmotic concentration of the surrounding fluid, the phenomenon is referable to molecular imbibition. But under normal conditions the cornea of the eye does not swell, and remains clear. Hence, in the living eye there must be some contrivance that controls the avidity of the cornea for water, and this regulator was discovered by *Leber* to be the endothelium.

This may be demonstrated by the following experiment: If we injure the endothelium on the posterior surface of the cornea of a rabbit in three places with a fine needle, the cornea begins directly to become cloudy at these places. As long as the endothelium at the points of injury is not regenerated, the aqueous continues pathologically to enter the cornea, which continues to swell and to become cloudy. To preserve the transparency of the cornea, it is the duty of the endothelium to prevent an abnormal entrance of water from the anterior chamber.

Whether this function of the endothelium is an expression of vital activity or of coarser physical properties of these cells, is as yet undecided. At all events the cornea of an enucleated eye will remain clear for several hours if the eye is protected from evaporation, as has been mentioned. We also know from the researches of Bullot, that when there is a lack of oxygen about the eye to be preserved, the endothelium of *Descemet's* membrane quickly breaks down and the cornea rapidly becomes opaque through the entrance of the aqueous.

On the other hand it must not be thought that the endothelium of *Descemet's* membrane absolutely prevents the passage of water into the cornea, for the cornea loses a certain amount of water through evaporation and, in the periphery, through the lymphatics and, even though the loss is small, it must be replaced. Hence, the function of the endothelium is regulative in character.

As the ground substance of the cornea has the property of absorbing water and as a regularly controlled imbibition of water must take place, we have to accept the theory that the cornea receives its nourishment primarily by way of diffusion. The question then arises: What do we know of the processes of diffusion in the cornea?

We continually make use of them. If atropin is dropped into the eye of our patient, the pupil dilates because by diffusion this substance passes through the intact cornea into the aqueous, where it is able to act upon the iris. Actual proof of the entrance of atropin into the anterior chamber is afforded by removing the aqueous from animals thus atropinized and producing with it mydriasis in other animals. Experiments in diffusion have been instituted with such substances as eserin, potassic iodide, potassic ferrocyanide and fluorescin. Such soluble substances also diffuse backward from the aqueous into the parenchyma of the cornea. Nevertheless an accurate conception of the processes of diffusion that take place in the cornea cannot be obtained unless it is made clear that the epithelium and endothelium materially obstruct the diffusion of such crystalloid substances. It may be readily shown that all these substances diffuse much more easily into the cornea when the epithelium is injured or removed. An experiment which is used a great deal in practice is to place a drop of a solution of fluorescin in an eye in which we suspect an injury of the epithelium of the cornea. The area from which the epithelium is abraded becomes deeply dyed immediately, while the large portion of the cornea covered by epithelium does not accept the stain.

E. v. Hippel has shown that endothelial defects also are rendered visible by the instillation of fluorescin into the conjunctival sac, but a satisfactory explanation of the fact that such endothelial defects are more weakly stained when the fluorescin enters the anterior chamber from the blood-stream is as yet lacking.

In any case the quantity of soluble substances that pass through the intact cornea by way of diffusion is very small. This is another evidence that the epithelium and endothelium of the cornea have the attribute of very greatly influencing and restricting diffusion. Everything indicates that within the epithelium and endothelium the cement substance furnishes the path for the diffusion throughout the substance of the cornea.

If, now, it is taken for granted that the nutrition of the cornea is derived chiefly from the anterior chamber, it becomes necessary to discover whether colloid substances can pass through *Descemet's* membrane as well as crystalloid. An evidence that they may is furnished by the fact that when, in severe injuries of the eye. a fluid hemorrhage remains for a long time in the anterior chamber, a peculiar clinical picture is produced by the seeping of a derivate of the coloring matter of the blood through the parenchyma of the cornea by way of diffusion.

An essential factor in nutrition for any organ, or any cell, is an elective capacity of its protoplasm. The cells of the cornea possess the power to take up into themselves certain substances and to oppose a powerful resistance to the entrance of others. This is apparent in every stained microscopic preparation, where the specific affinity of certain parts of the cell to various stains is evident. In vivo this elective behavior of the cells of the cornea is even more striking. This will suffice concerning the nutrition and metabolism of the cornea.

We will now bring in our first patient and study a normal cornea in vivo. It forms the transparent portion of the capsule of the eyeball. Its horizontal diameter is somewhat greater than its vertical, so it is not circular in form, but elliptic. As the arch of its curvature is greater than that of the sclera, there is a shallow groove at the limbus where the cornea and the sclera unite. Its horizontal diameter is about 11 mm., its vertical about 10 mm. It is thinner in the centre than at the margin, where its thickness is only about 1 mm. The conjunctiva scleræ overlaps the cornea a little and then continues as corneal epithelium.

The points to be considered in an examination of the cornea may be included under three headings:

1. Form, size, and curvature of the cornea. Great deviations from the normal will be met with in these respects.

2. Surface and sensibility. Normally, the surface is smooth and lustrous, each epithelial cell lying close to the others. It is also very sensitive. This may be shown by gently touching it with a bit of cotton, carefully avoiding the lashes and the margins of the lids, when a reflex closure of the lids will be produced, and the eye will begin to lacrymate. Later, cases will be seen in which the surface of the cornea is no longer normal and lustrous and in which its sensibility is largely or totally lost.

3. The transparency of the cornea interests us more than anything else. It may be taken as axiomatic that the greater number of morbid disturbances in the cornea attract attention first through the decrease of the transparency; this explains why the physician and the ophthalmologist diagnose most diseases of the cornea from the sort of obscuration present. We need, therefore, to study opacities of the cornea more accurately.

First we need to know by what methods of investigation we may learn concerning these three important points and the deviations from the normal.

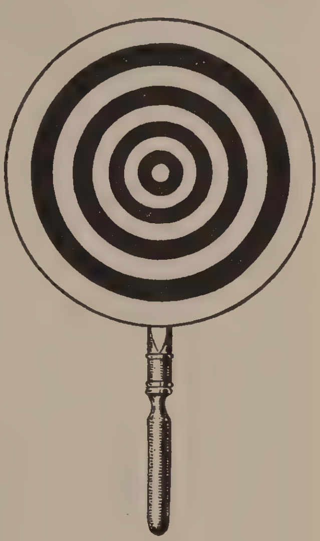

Fig. 5.—*Placido's* Keratoscope.

To begin, we examine the eyes of our patient by daylight. The iris may be seen through the normally clear cornea, the mirrorlike lustre of which is particularly striking. If we place the patient facing a window a vivid reflection of the latter appears on the cornea, which serves as a convex mirror with a surface of a rotation ellipsoid, for its curvature is somewhat flatter at the periphery than at the centre. This little convex mirror reflects a beautifully clear, regular, upright fac-simile in miniature of the opposite window, and it is this reflection from the cornea that gives the eye its brilliancy and fire.

This mirroring characteristic is taken advantage of in the study of the form, surface and lustre of the cornea. Every irregularity of surface, even when the epithelium is present, every morbid change in the epithelium, every absence of epithelium, every loss of smoothness of the cornea becomes immediately perceptible from the distortions, breaks of continuity and irregularities to be seen in this little reflected image. We utilize this mirroring of the cornea by means of an instrument known as the keratoscope of *Placido*, or Placido's disc, consisting of a disc with concentric black and white rings. If this disc is held so as to be mirrored on the cornea, the delicate reflection of the rings may be seen. Not only the centre, but the whole of the cornea may be examined in this way by having the patient look in various directions. It will be seen that the reflection of the disc from the centre of the cornea is perfectly circular, while in the peripheral portions it is drawn out longitudinally, because there the surface is flatter. The neophyte should not consider this anything pathologic. Invariably, when the cornea is normal, this drawn out reflection of the disc is clean cut in its outline. The case is quite different when the surface of the cornea is abnormal.

The third, and for the general practitioner the most important method of examining the cornea, is the focal, or oblique illumination. The patient is seated in a darkened room near a lamp, so that the source of light is at his side and somewhat ahead of him at a distance of about ¾ m. We then focus the rays of light from the flame of the lamp on the cornea with a convex lens of 20 dioptres. This method is called focal illumination because the area of the cornea to be examined is brought into the focus of the lens. This point stands out with extreme distinctness for two reasons: first, because it is intensely illuminated by the concentration of the cone of light; secondly, because the circumjacent parts remain in darkness. This sharp contrast permits the revelation of slight disturbances which could not be perceived in any other manner. When this method is properly employed we may illuminate each part of the cornea in turn, we may plunge the focus to the deeper parts and examine the anterior chamber, the iris, the lens and even the anterior portion of the vitreous. We shall employ this method daily, and students should become proficient in its use.

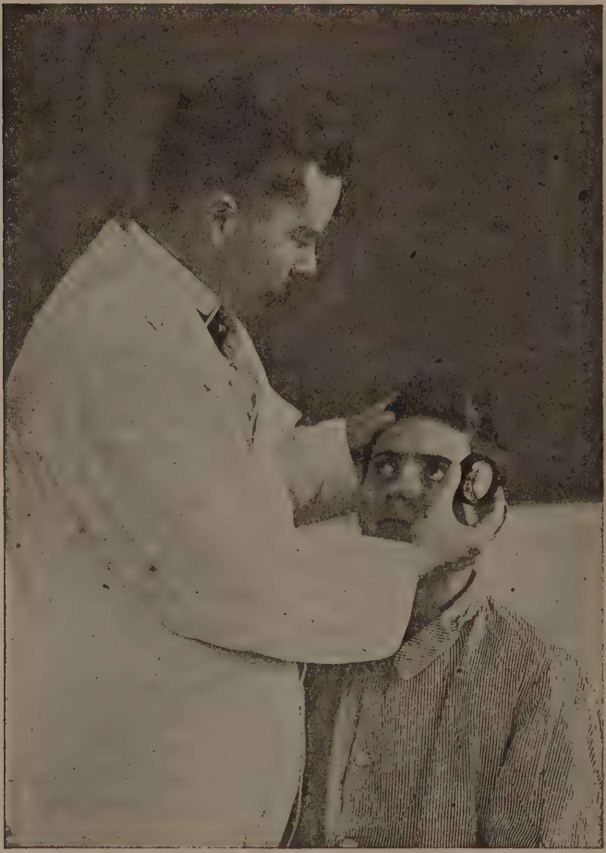

Fig. 6.—Focal Illumination.

Focal illumination shows that the normal cornea is not absolutely transparent, but reflects some light, for the area illuminated has apparently a gray tone. This reflection is understood at once as soon as we think of the anatomical construction of this tissue. Our wonder is rather excited that such transparency can be attained in this dense structure. This method of focal illumination may be used to still better advantage if the eye is viewed through another convex lens, held in the other hand, while the cone of light is thrown on the eye with the first lens, or loupe. This simple manœuvre is very useful in practice. By its means we can discover very slight erosions of the cornea and the minutest foreign bodies, and can also study other morbid changes throughout the anterior segment of the eyeball.

Here, in the clinic, we employ for all these examinations in the anterior segment of the eye a very superior optical instrument, *Zeiss*' corneal microscope. This loupe gives us not only clear, well-illuminated pictures, but also, as it is a binocular instrument, binocular perception of depth. By proper manipulation of its adjustments we can examine in succession the surface of the cornea, the parenchyma, the posterior surface, the anterior chamber, the iris and the lens. By means of this loupe we are able also to see the circulation of the blood in the conjunctival vessels and in the newly formed corneal vessels, a phenomenon which hitherto has probably been observed only in the physiological study of the frog.

Finally, we use the ophthalmoscope for examination of the cornea, the rays of light from which pass through its tissue. That important information is sometimes obtained in this manner, will be demonstrated in our clinical work. These methods of examining the cornea need practice: its mirroring, keratoscopy, focal illumination, observation with *Zeiss*' loupe, and examination with the ophthalmoscope.

If the normal corneas of different patients are compared, it will be found that those of the older patients reflect light more than those of the younger ones. From this it may be concluded that a certain increase of density takes place physiologically in the corneal tissue as age approaches. A true physiological opacity appears in many old people, that is called the arcus senilis of the cornea. In this elderly patient, who has an otherwise clear cornea, there is a narrow, white-gray line about 2 mm. broad that runs concentrically with the margin of the cornea in the parenchyma near the limbus. It does not adjoin the limbus, but is separated from it by a perfectly clear zone. The opacity itself is sharply marked on the side toward the margin of the cornea, while on the side toward the centre it blends gradually with the clear tissue. In this case the opacity forms a closed ring, but in its evolution a gray arc appears first above, then below, and finally these unite.

Anatomically, we find deposits of hyaline flakes and of lime in the region of the opacity, together with lipoid particles. The peripheral position of the arcus senilis renders it harmless to vision, but sometimes it invalidates somewhat the result of an optic iridectomy.

3. Clinical Examination of the Normal Iris and the Cardinal Signs of Primary Iritis and Iridocyclitis

As we pass more deeply into the eye, we come to the iris. It is preconditional to the study of the morbid conditions of this membrane that its normal structure and clinical appearance should first be understood, so we will briefly review its clinical anatomy.

The iris is stretched in front of the lens, and may be compared to the diaphragm of a photographic apparatus. It divides the anterior segment of the eyeball into two parts which communicate through the pupil, the anterior chamber, which is the larger, and the posterior chamber.

It consists of a loose, spongy tissue which contains, in addition to numerous, mainly radiating, bloodvessels, as its most essential components, the two muscles, the sphincter and the dilatator pupillæ. Its layers from before backward are:

1. The anterior layer of stroma. This covers the entire surface of the iris

and contains in particular numerous anastomosing pigment cells, the chromatophores. An endothelial coat over the surface is lacking.

2. The peculiar vascular layer. The vessels lie in a wide-meshed, loose connective tissue.

3. Behind the vessels lies the delicate dilatator, in front of which, and adjoining it toward the pupil, is the more powerful sphincter.

4. The posterior layer is represented by the deeply pigmented epithelia of the retinal pigment layer, which, folding over anteriorly, edges the margin of the pupil with black.

The most marked phenomenon of the iris in the living eye is its *variety of colors.* The attributes sung by the poet, and those that fire the amorous swain, lie chiefly in the hue of the iris.

The color of the iris depends essentially on the pigment contained in the connective tissue layers, both in the anterior layer of stroma and in the deeper layers, and stored up in the branching cells, the chromatophores. Another form of pigment in the iris is the retinal pigment, which fills the cells of the retinal posterior layer of epithelium. In all eyes this coat of retinal epithelium is richly provided with pigment, while the quantity of pigment in the stroma is extremely variable, and the color of the iris, whatever it may be, is dependent on the ratio between these two kinds of pigmentation.

In the first type the iris is blue. This color is only a physical phenomenon. In such persons the iris is delicate and very poor in pigment, so that this unpigmented iris tissue forms only a cloudy layer over the brown, strongly pigmented layer of retinal pigment common to all eyes. This dark background of brown epithelium shimmers through the rather cloudy medium of the iris tissue and appears bluish under the optical conditions present. The long waved rays of ordinary daylight are absorbed by the iris in greater quantity than the shorter waved blue and violet rays, more of which are reflected. A dark background seen through a hazy medium appears bluish, just as the veins of the hand seen through the skin appear to be blue. The eyes of infants are very often blue because the irides are delicate and slightly pigmented, but in the course of life the tissues of the iris become denser, tenser and richer in fibres; blue eyes frequently become gray.

In the second type the color is light gray. In it are clearer spots, non-pigmented places, where the anterior layer of stroma is thickened.

Other colors range from the brightest yellow to the deepest brown. The greater the quantity of pigment contained in the stroma, especially in the anterior layer of stroma, the more does the surface of the iris have a slightly dusty and granular appearance. The vessels are then surrounded by tissue containing coloring matter, and the chromatophores are overfilled with pigment. Many cells containing pigment are also to be found in the deeper layers of the iris.

In another case we see a sort of pigmentation in spots, which is called nævus of the iris. These islandlike heaps of pigment often appear as dark rust colored, or black specks in a gray or blue iris. The iris of one eye also may be brown, that of the other blue. The color of the iris, however, is in harmony with the pigmentation of the rest of the body.

Passing now from simple observation to focal illumination, each observer will be confronted with another normal phenomenon, an elegant marking of the iris, formed by the elevations and depressions on its anterior surface, and known as the ''relief'' of the iris. This needs to be closely examined. It is best marked when the iris is non-pigmented.

This relief is formed of numerous, rather prominent, radiating cords, which are the larger bloodvessels coursing in the stroma. A part of these radiating vessels bend almost at a right angle at the junction of the inner and middle thirds of the iris and run concentrically to the pupil. These lines and curves, composed of both arteries and veins, form a wreathlike design, which marks at the same time the thickest and most elevated region of the iris, called by *Krueckmann* the "ruff" of the iris.[1] This divides the iris into two unequal areas, the pupillary and the ciliary zones. To its inner side lies the pupillary zone, in which the brown edge of epithelium may be seen at the margin of the pupil. This zone is often of a color different from that of the ciliary zone.

I will now place the patient in front of *Zeiss*' loupe, by means of which the sphincter can be distinctly seen shimmering through the non-pigmented iris. It is a flat band of smooth muscle fibres and lies next to the posterior surface. Its presence is marvellously demonstrated by the continuous play of motion in the pupil, which causes a change in its pigmented edge. When, by alternate shading and illumination, the pupil is contracted, the edge of pigment becomes broader; when the pupil dilates, it becomes narrower. When the sphincter contracts strongly, slight tremors may sometimes be seen in the peripheral portions of the iris.

We learn from embryology that the brown pigmented edge of the pupil and of the sphincter are epithelial descendants of the secondary optic vesicle. They are the only terminal prolongations of the entire central nervous system which we can perceive without artificial aid.

To the outer side of the "ruff" of the iris lies the ciliary region. This contains the greater part of the radiating vessels, and in it the "relief" is best observed.

Between the radiating lines lie a number of deep, sharply defined places, the crypts or lacunæ of the iris. The endothelial coat of the anterior surface is wanting in these places, so they are looked upon as openings that lead into the spaces in the inner tissue of the iris. It is very possible that this arrangement contributes in favor of the rapid changes in volume of the iris as the pupil contracts and dilates.

I will also call attention to the furrows visible on the anterior surface, which run concentrically to the margin of the cornea in the ciliary portion of the iris. These are known as contraction furrows, and are caused by the folding of the iris during the play of the pupil. The depressions between these folds, into which the anterior surface is thrown during dilatation of the pupil, are the furrows. They contain little pigment, and can be seen most clearly when the iris is dark and the pupil contracted.

Finally, there is a certain lustre of the normal iris, due to the wealth of protoplasm in the anterior layer of the stroma. This is most marked when the iris is non-pigmented. When there is much pigment this appearance is lacking, and the iris looks, as already mentioned, more granular, or dust strewn, but its outlines are always sharp and clear.

Inflammations stand preeminent among all the diseases of the iris. We shall not begin, however, by bringing forward the various forms of iritis according to their etiology, for that is properly learned only from the observation of many cases. The cardinal symptoms, which are more or less marked in every iritis, should first be learned and understood.

An inflammation of the iris may be due to morbid conditions in other parts of the eye, as, for example, the iritis that accompanies ulcus serpens, diseases of the conjunctiva, or of the internal parts, such as abscess of the vitreous. In such cases the iritis is secondary. To-day we are interested only in primary iritis.

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[1] Also known as the lesser circle of the iris, *F.*

Of the well-known symptoms of inflammation—rubor, tumor, dolor, and calor—the pain in an iritis is an extremely variable and uncertain symptom, and the sensation of heat is rarely complained of; therefore we are primarily concerned with the other two phenomena.

We find in the eye of this patient a well-marked pericorneal, or ciliary injection; the pupil is smaller than that of the other eye and reacts very slowly, if at all, to brilliant oblique illumination. All of these symptoms are due to the increased hyperæmia. More closely examined, the radial vessels are seen to be dilated. The iris is supplied with blood by the ciliary vessels of the eye. When an inflammation takes place in this vascular region, hyperæmia is a necessary consequence, and this causes a distention of the iris and a contraction of the pupil. In addition to this the sphincter becomes more strongly contracted by the circle of inflammation. Thus the first cardinal symptom of iritis, the ciliary injection and contraction of the pupil, is explained.

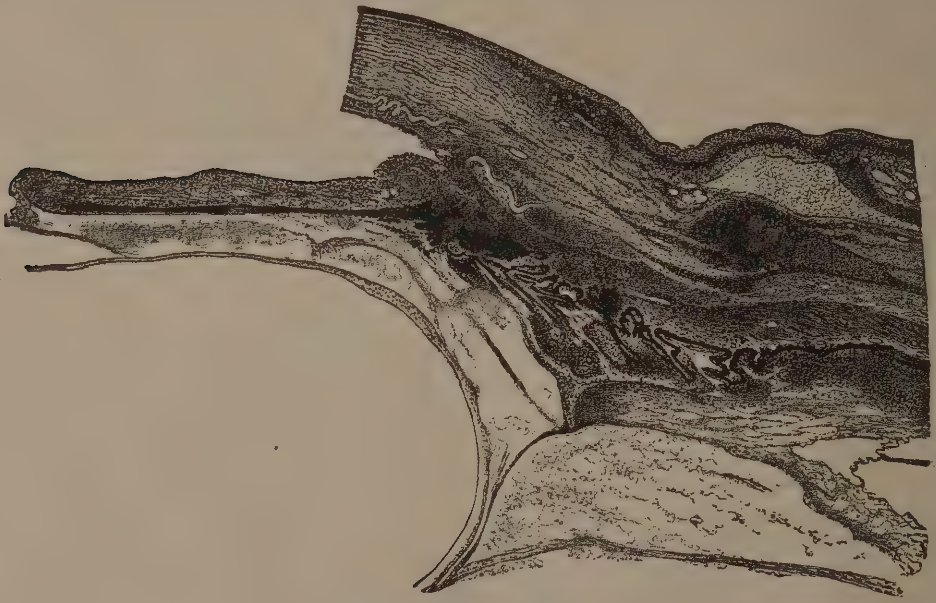

FIG. 7.—Iridocyclitis.

This is not all. When the two eyes are compared, a considerable *discoloration* of the iris is to be observed. In the healthy eye of our patient, the iris is blue; while in the other it is distinctly greenish. Likewise, when the normal color of the iris is gray, the greenish hue is to be distinctly seen in inflammation; with a brown iris the discoloration is clinically less pronounced. The iris has lost its lustre. This is because of a cloudy swelling of the anterior layer of stroma, the outer surface of the iris which is rich in protoplasm. Discoloration and loss of lustre are also due to the hyperæmia, though the next cardinal symptom, the inflammatory exudation, likewise plays a part.

Inflammatory hyperæmia is accompanied by an increased permeability of the walls of the vessels, and by the escape of an inflammatory exudate.

This exudation causes the next and most important symptom in any iritis: the obscuration of its beautiful markings. In this patient the "relief" of the iris and the structure of its surface appear to be effaced in large measure. This is the result of the exudation in the iris. Because of this the iris is more voluminous, the pupil more contracted and its mobility limited.

The final indication of iritis is the formation of this exudate.

An exudate may be composed, 1, of a serous, very albuminous fluid, the inflammatory œdema; 2, of threadlike coagulations, fibrin; 3, of exudation cells, pus corpuscles. According to the presence or preponderance of these individual constituents, we speak of an iritis as serous, serofibrinous, fibrinous, fibrino-purulent, or purulent. Hence, if the physician suspects an iritis, the first clinical question to be answered is: To what form of inflammation does this iritis belong? The answer is easy if we examine the exudate somewhat more carefully.

The iris lies stretched between the anterior and posterior chambers, so there are three possible places where the exudation may have to be sought—in the iris itself, in the anterior chamber, and in the posterior chamber. In this case the obscuration of the details of the iris have been noted, therefore a serofibrinous, or fibrinous exudate is present in the tissue of the iris. With the loupe it may be seen that the anterior surface is covered with fibrin, which contributes materially to its washed-out appearance, while the iris itself is thickened by the exudation in its inner layers.

Such an exudate is by no means confined to the iris. As the excretion increases, the fibrinous exudate may extend from the margin of the pupil to the anterior capsule and occlude the pupil with a dense membrane, a condition that is known as occlusio pupillæ. In this case the entire pupil looks gray. In obstinate cases this fibrinous occlusion of the pupil persists, the fibrin becomes organized and replaced by connective tissue. The name applied to this in German is Pupillenschwarte.

Thus we have already learned one consequence of iritis, for it is evident that such a fibrinous membrane covering the pupil will interfere gravely with vision.

The exudate from an inflamed iris may also be deposited in the posterior chamber, with a certain predilection for the place of contact between the margin of the pupil and the anterior surface of the lens, and lead to the formation of adhesions between the margin of the pupil and the anterior capsule, which we call *posterior synechiæ.* At first these are fibrinous, but often become more firm and solid. A physician must always be on the lookout for these posterior synechiæ in every case of iritis. They are present in this case. We may differentiate them into two principal varieties. If the adhesion is confined to the retinal pigment layer it is usually linear, or seamlike (marginal), and then black, or gray-brown adhesions of the edge of the pupil to the capsule of the lens may be seen in various places. But if a greater extent of the posterior surface of the iris is adherent to the lens at such a place, single circumscribed foci of inflammation are present, which cause broader, or serrated adhesions. It is not difficult to recognize the posterior synechiæ in the present case, in which they have already become distinctly formed, but in an acute attack with great contraction of the pupil they may sometimes be overlooked. We have a simple means of determining in any case of iritis whether such synechiæ are present or not. We place a drop of atropin in the conjunctival sac; the pupil begins to dilate, but is held fast at the points of adhesion so that the posterior synechiæ come into view as points, dentations, or seams. In grave cases the posterior synechiæ may be so massive and extensive that the entire pupillary margin of the iris becomes adherent to the anterior capsule of the lens, producing the condition known as seclusion, or exclusion, of the pupil. This is usually associated with a total or partial occlusion as well. The peripheral and middle portions of the iris are bulged forward, producing the condition known as crater pupil.[1]

The genesis of this morbid condition is readily understood if we recall that seclusio pupillæ shuts off communication between the posterior and anterior chambers, thereby preventing the aqueous from passing through the pupil into the anterior chamber and damming it up in the posterior chamber, where it presses the iris forward. In such cases the ciliary body participates in the inflammation, which produces an accumulation of inflammatory œdema in the posterior chamber that aids in pushing the iris forward. The protrusion forward of the peripheral portion of the iris diminishes the angle of the anterior chamber, and the accumulation of fluid behind it leads to an increase of the intraocular pressure and to excavation of the optic nerve. This is another consequence of the formation of exudates in this disease. The tension must be tested in all cases of iritis.

In order to determine whether seclusion is present, atropin is instilled and the tension tested. It not infrequently happens that the pupil dilates in one or more minute places, the narrow openings sufficing to prevent increase of tension. If these consequences of exudative formations in iritis are competent to excite apprehension, the following are much more so.

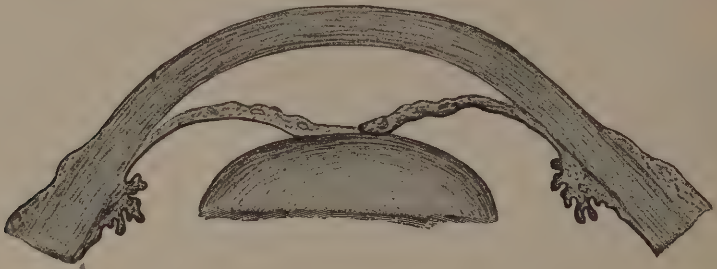

FIG. 8.—Crater Pupil, Due to Seclusio Pupillæ.

I wish here to impress a fact to be remembered; that in the vast majority of cases of iritis, the ciliary body is involved, and that the iritis is actually an iridocyclitis. The reason for this is readily understood, because the iris issues from the ciliary body. When iridocyclitis is present, the ciliary body is frequently sensitive to pressure. If the ciliary body excretes an abundant fibrinous exudate the posterior chamber may be completely filled with fibrin, and then not only the margin of the pupil, but the entire posterior surface of the iris becomes adherent to the lens. This condition is known as *total posterior synechia.*

We recognize this condition by the fact that the iris, including its peripheral portion, is drawn backward by the shrinkage of the masses of fibrin in the posterior chamber during organization and transformation into connective tissue.

Exudates from the ciliary body may very easily take place into the vitreous, where they act optically as opacities and induce considerable visual disturbance. These opacities of the vitreous in iridocyclitis have another, still more serious, signification. They may become organized through granulation tissue. The tissue of the vitreous is in part resorbed, but the granulation tissue may proliferate, often involving the connective tissue of the head of the optic nerve, then it shrinks and detaches the retina. Ablatio retinæ may thus be the termination of a grave iridocyclitis.

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[1] The German term Napfkuchenform der Iris, cup-cake form of the iris, would hardly be intelligible to American readers, as their cup-cakes differ in shape from the German. The condition is known to French writers as "iris bombe," to English and American as "funnel pupil" and "crater pupil."—F.

These vitreous exudates sometimes completely envelop the lens, coming from both the posterior chamber and the vitreous, and the access of nutritive fluid to the lens is thereby destroyed. The lens begins to grow opaque and cataracta complicata develops, another consequence of a serious cyclitis.

Such eyes usually shrivel, as the result of the destruction of the vitreous, and the final act in this tragedy is atrophia bulbi. Such results may not only be studied in specimens, but will be met with in patients.

We will now turn from these exudates in the posterior chamber to the anterior segment of the globe, for it is self-evident that in iritis exudates may form in the anterior chamber as well. We must indeed look for them in every case.

Such an exudate is the hypopyon in ulcus serpens. When there is pus in the anterior chamber a purulent iritis is naturally present, while we have thus far been dealing with fibrinous exudates only in the iris and the posterior chamber. The purulent, or fibrino-purulent, exudate in the anterior chamber tends to sink in the aqueous to the deepest part of the chamber, where its upper surface forms a horizontal line.

Another form of exudate, the so-called precipitates, are much more common in primary iritis. In the patient now before you, we find a great number of minute gray specks arranged peculiarly on the posterior surface of the cornea. By simple focal illumination, or with the aid of a lens, or still better with *Zeiss*' loupe, it may be seen that the largest deposits lie deepest on the posterior surface of the cornea and that the arrangement comes almost to a point. In other cases the arrangement is irregular.

We have two ways of proving that these formations lie on the posterior surface of the cornea. First, by means of the binocular *Zeiss*' loupe they may be located very definitely. Second, when the anterior chamber is opened; in such cases, we see a great part of these precipitates suddenly washed out of the eye by the escaping aqueous.

In cases of iritis characterized by the presence of these precipitates, there is but little inflammatory exudation, and the condition is called iritis serosa. There is only a slight migration of exudate cells and excretion of fibrin. The cells become glued together into little lumps, which, suspended in the aqueous, are thrown by the movements of the eye against the posterior surface of the cornea, to which they adhere—the heaviest and largest lumps at the bottom. Frequently they can be found only by a careful search. Examination of these deposits with the loupe shows that they are of a gray, or yellowish-gray color, especially when the iris is blue or gray. But they may contain pigment, which is a sign that particles of pigment from the epithelium of the retinal pigment layer, or from the ciliary body, have been swept away by the round cells. If these precipitates alone are formed, the clinical diagnosis is serous iritis; but if there are, in addition, fibrinous exudates in the iris and posterior chamber, we speak of a serofibrinous iritis.

We need still to be able to differentiate these precipitates from opacities in the posterior layers of the cornea. Precipitates lie uniformly at the level of the posterior surface of the cornea and are sharply distinguished from their environment. Opacities in the posterior layers of the cornea usually lie in different planes and blend gradually with the surrounding parts. This differentiation is not always easy, for little opacities in the posterior layers of the cornea may arise from the precipitates in an iritis and produce a circumscribed dulness of the opposite surface epithelium. This is a secondary keratitis.

4. The Normal Lens in Youth and Old Age

Prior to the consideration of the diseases of the lens, one of the most important and interesting organs of the eye, we must study the clinical anatomy and the clinical picture presented by the normal lens.

This organ, named because of its lenticular form, lies behind the iris within the circle formed by the ciliary processes. It separates the anterior from the posterior segment of the globe, the iris with its pupillary margin lying upon its *anterior surface*, while its *posterior surface* rests in the fossa patellaris of the vitreous. The centre of its anterior surface is called the anterior pole; that of the posterior surface the posterior pole; the line joining the two poles is the sagittal diameter of the lens, the lenticular axis. The equator of the lens is situated where the anterior surface bends over into the posterior. The diameter of the circle formed by the edge of the lens is termed the equatorial diameter; the equatorial plane falls somewhat to the rear of the tips of the ciliary processes. The distance from the anterior pole of the lens to the posterior surface of the cornea is about 2.6 mm.

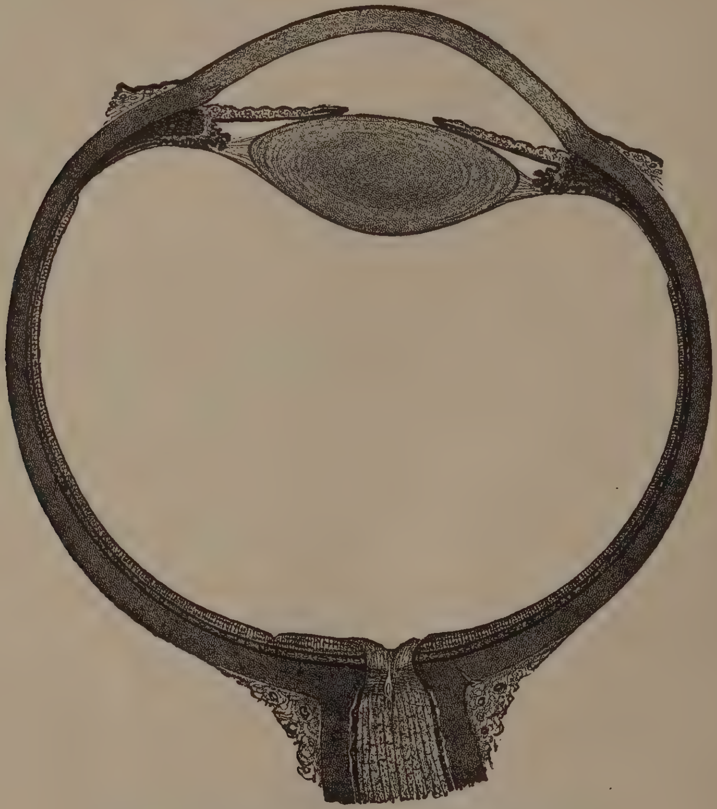


FIG. 9.—Section Through a Normal Human Eye.

The lens is composed of 1, the lenticular capsule; 2, the lenticular epithelium; 3, the lenticular fibres. The capsule is a fine, homogeneous, transparent membrane, which is thicker in front (about 0.007 mm.) than behind (about 0.002 mm.), and very elastic, as is frequently demonstrated when it rolls up after an injury. The fibres of the zonule of *Zinn*, the suspensory ligament of the lens, blend with its most superficial layers. The fibres of the zonule come from the inner surface of the ciliary body through the ora serrata, are closely connected with the pars ciliaris of the retina, leave the ciliary body at the tips of the ciliary processes, divide into fibrils and stretch over to the lens. The fibres of the zonula usually divide into three portions, one of which is inserted at the equator, one in front of it and one behind it.

Between the equator of the lens and the bands of fibrils from the zonula, there appears on section a triangular space, the so-called canal of *Petit*. This is not a closed space, but is in communication with the posterior and vitreous chambers.

Returning to the capsule of the lens, we find that during life it thickens, and in suitable preparations its construction of fine lamellæ can be recognized. This brings us to its genesis.

The embryology and pathology of the lens show that its capsule is a product of its epithelia. It has been calculated from the growth of the lens, that its capsule, in an adult, covers an area approximately two-thirds greater than that of the surface of the lens in an infant. If the capsule of the lens became enlarged by being stretched, it would become thin; but, in fact, its thickness increases throughout life. The marvellous uniformity of the capsule of the lens is an evidence of the constructive ability of the lenticular epithelia.

The epithelium of the capsule is found beneath the anterior capsule; it forms a single layer of cubical cells which ends with a sharp margin at the equator. The average diameter of an epithelium is about $\frac{1}{50}$ mm., so it may be calculated that there are approximately half a million epithelial cells that cover the anterior capsule, provide for the formation of the latter, and care for the development of the lens fibres.

With *Rabl* we divide the lens fibres into three groups, the central, the transitional and the principal fibres. The central fibres are the oldest and form the nucleus. Their nuclei have been lost and they appear on section to be dentate and shrivelled. The transitional and principal fibres compose the cortex of the lens. They are from 7 to 10 mm. long, hexagonal in section, and they grow toward the poles of the lens. As the capsular cells at the equator are arranged in meridional rows it follows that the fibres form radial lamellæ, which compose the lens like the different sectors of an orange. This sectorlike arrangement may be demonstrated experimentally by soaking the lens of an animal in a weak solution of potash, after which it may be peeled off in sectors. This sectorlike arrangement is often evident in diseases of the lens.

Finally, a word concerning the stellate radiations of the radii lentis. The fibres of the radial lamellæ do not unite in a point at the poles, but gather in radiating lines which form a stellate figure about the pole.

With these preliminary remarks upon the anatomy we turn to the vital peculiarities of the lens, in which throughout life, under purely physiological conditions, a number of clinically interesting changes take place, with which the physician must be acquainted if he would distinguish the normal from the abnormal, the healthy from the morbid lens. The cause of these changes we find in growth and age. Senility in particular, which produces so many visible changes in our organism, leaves its traces even in the individual organs. In the eye it is the lens in which senile changes are particularly marked. We will therefore, to-day, compare the normal young and the normal old lens.

As long as the lens is healthy it remains transparent in youth and old age, though, as we shall learn when using the ophthalmoscope, the transparency grows less with age. The normal senile lens is differentiated from that of youth by four characteristics: 1, the size; 2, the form; 3, the consistency; 4, the color. These differences are readily demonstrated with animal lenses, some of which have been preserved, some freshly prepared. After these have been studied we may turn to the examination of the lens in the living eye, and see in how far our clinical methods of investigation enable us to differentiate between old and young lenses.

The lens is one of the organs of the body that continue to grow even in extreme old age, like the nails and the hair. The meridional epithelial cells of the equator give rise to new fibres in the lens throughout life. This is accompanied by a diminution of the water and a condensation of the interior of the lens, as may be both seen and felt, despite which the increased volume from this steady growth, due to apposition of new fibres, is so great that, according to *Priestley Smith*, the volume and weight of the human lens increases one-third, the diameter one-tenth, between the ages of twenty-five and sixty-five. The difference in size will be recognized immediately on examination of freshly prepared lenses from calves and from oxen. A comparison of the preserved lenses of children and of adults is likewise convincing. In the following table, after *Priestley Smith*, may be seen the increase in weight and volume of the lens with advancing age.

WEIGHT AND VOLUME OF THE HUMAN LENS WITH ADVANCING AGE, ACCORDING TO PRIESTLEY SMITH.

Age in Years.	Average Weight in mg.	Average Volume in mm.
20–29	174	163
30–39	192	177
40–49	204	188
50–59	221	205
60–69	240	225
70–79	245	227
80–88	266	244

The average weight of the human lens in middle life is about 0.22 gram. The senile lens is, however, not only larger and more voluminous, but of a different form. A lens freshly dissected from the eye of an infant is nearly globular; a senile lens has a much greater equatorial than sagittal diameter, and its posterior surface is much more arched than its anterior. In the infant the equatorial diameter is about 6 mm., its thickness about 4.5 mm.; the equatorial diameter of the senile lens is about 10 mm., the sagittal between 4 and 5 mm.

The cause of this change in the form of the growing lens has not yet been ascertained, but it is thought that the constant tension of the zonula takes a part in its production. At all events, in cases in which the zonula is partially wanting, the lens may have a different form at the places where this tension is gone.

The third differential characteristic between young and old lenses, the consistence, is very distinct, as may be demonstrated by taking the lenses of calves and of oxen between the fingers. The first are felt to be quite soft, while the latter, on the contrary, are felt to have in the centre a firm, very glutinous mass, which cannot be crushed by digital pressure, though the softer cortical layers are easily rubbed off. This can be recognized just as clearly in the human lens. The hardening of the lenticular nucleus is due to a process to which we apply the name sclerosis. This begins in youth and advances steadily to the most extreme old age, with the result that the oldest lens fibres continually become harder and harder from dehydration and flattened.

This process of sclerosis goes hand in hand with an increase of the refractive index, caused by the dehydration, as *Halben* pointed out; but, whether the increase of the index in the human lens is due wholly to the loss of water, or, in part, to chemical changes, is still uncertain. The latter theory seems to me the more probable.

A firm nucleus thus develops within the lens. It is clearly demonstrable about the twenty-fifth year, increases in size at the expense of the soft cortex as age advances, until, finally, in extreme old age, the lens consists almost wholly of a hard nucleus. This development of the nucleus is made clearly visible when a senile lens is divided in half, when not rarely the nuclear portion appears dry, and distinct from the moist and lustrous cortex. The significance of this process of sclerosis with regard to the function of the eye will appear presently.

The final characteristic by which young and old lenses may be distinguished immediately is a striking contrast in color. First I will call attention to the fact that the animal lenses thus far considered are colorless. We have here the lens of a monkey, which is distinctly yellow. Even the perfectly normal lens of an infant is not colorless, but yellowish, as may be seen by placing it on a sheet of white paper. By this means human lenses may be differentiated from the lenses of animals, with the exception of monkeys and apes. This yellow hue of the human lens constantly becomes more marked as age advances. Generally the color of the healthy lens varies from a clear wine yellow to a deep brown, with extremely great individual differences, and regularly decreases from the nucleus toward the periphery. In some cases the brown color of the lens becomes so great, and the absorption of light thereby induced so disturbing, that we are obliged to remove the lens from the eye. The causes of all these differences in consistence and color between young and old lenses are still unknown.

We will now turn to the living eye and study the healthy lens. There are three principal methods at our disposal for the usual clinical examination of the lens: 1, ordinary observation by daylight; 2, focal illumination; 3, ophthalmoscopic observation. With these we will compare the lenses of a child and of an elderly person. In the child the pupil appears black in ordinary daylight, and nothing in particular can be seen of the lens. By focal illumination in a darkened room the pupil has a delicate gray sheen. This is strikingly distinct when comparison is made with the pupil of an eye from which the lens has been extracted, which is deep black when examined in this manner. This shows that the healthy, young, perfectly normal and transparent lens reflects some light, which is not surprising when we consider the anatomical construction of the lens from a great number of fibres.

The room is now made perfectly dark, and a light is moved about at some distance from the eye. By looking carefully, not only the familiar reflex from the cornea may be seen, but two other reflexes as well, one from the anterior and the other from the posterior surface of the lens. These are the *Purkinje-Sanson's* reflex images. The anterior surface of the lens acts, like the cornea, as a convex mirror.

The anterior lenticular reflex originates in the focal point of the reflecting surface, which, with a radius of 10 mm., lies 5 mm. behind the anterior and 1 mm. behind the posterior surface of the lens. The anterior lenticular reflex is an upright, small, virtual image, very faint, and usually seen with more difficulty than the posterior lenticular reflex. The dimness of the anterior lenticular reflex is largely due to the fact that it is not produced wholly by reflection from the anterior capsule, but partially by light reflected from the most anterior parts of the lens cortex.

In demonstrating the anterior lenticular reflex the patient is told to gaze directly forward, the light is held on his temporal side about 45° from the visual line, while the observer looks into the eye from the nasal side, likewise at an angle of about 45°. The light is then moved and the dim upright image is seen to move in the same direction.

The study of the anterior lenticular reflex with *Zeiss* loupe is interesting. If we direct it to the anterior surface of the lens at the margin of the pupil, the anterior reflex appears as a dimly luminous spot in which we can perceive numerous small, delicate, roundish areas. This delicate, shagreened appearance is plainly due to the capsular epithelium; for, in animal lenses in vitro, it is visible only on the anterior surface.

The posterior lenticular reflex can be demonstrated much more easily. It is cast by the concave posterior surface of the lens and is an inverted, small, real image. It lies in the focal point of the posterior surface of the lens, about 3 mm. in front of the latter, and about 1 mm. behind the anterior surface of the lens. If we now lower the light, the little shining point rises, while the corneal reflex moves in the same direction as the light. This form of examination is much employed when it is necessary to prove whether an eye contains a lens.

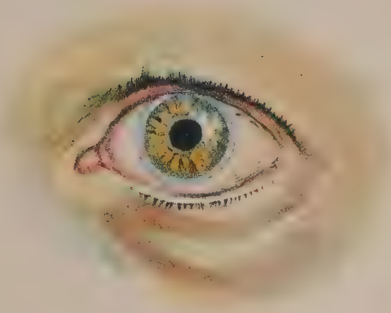
With the ophthalmoscope, we find that the young normal lens is perfectly transparent. Now and then we find in children a sharply circumscribed dark spot in the region of the posterior pole, an innocuous residuum of the atrophied arteria hyaloidea; with this exception nothing is to be seen of the lens itself. But it is this transparency and absence of opacities that shows us we are dealing with a normal lens. Our ordinary methods afford us little information concerning the form, color, size and consistence of the young lens. Its slightly yellowish hue cannot be perceived against the red background and we can determine nothing in regard to its form and size. I wish to call particular attention to the fact that even when the pupil is dilated the margin of the lens remains invisible.

In order to exhibit this margin, I have here a patient on whom an iridectomy has been performed. When the eye is turned far upward, the margin of the lens may be seen with the ophthalmoscope as a dark line. This is not always, even in the healthy eye, a smooth circular line, but may be slightly wavy, from an alternation of minute elevations and depressions. *Hess* has shown that these elevations are often more distinct after the use of atropin in the eye than after the use of eserin. Probably they are due to tension on the part of the zonula.

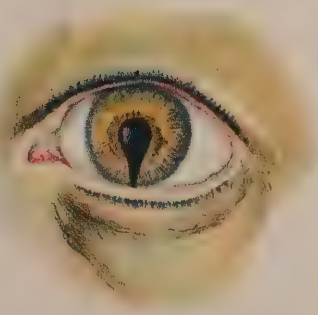
It can be proved only indirectly that this young lens is of the normal consistence. If we tell the child to read and hold the print very close to the eyes the accommodation functionates promptly. The lens is still capable of a great change of form. The result will be different when our second patient, with a senile lens, is examined.

The pupils of this elderly patient are much more distinctly gray even by ordinary daylight. Compared with the lens in youth the senile organ has far greater powers of reflection, and this physiologic lenticular reflex must never be confounded with cataract. With the ophthalmoscope the lens is seen to be transparent.

By oblique illumination we find in addition to the gray appearance, a yellow-



a Arcus senilis (gerontoxon) of the cornea.



b Congenital iridic coloboma (fissure).

Rebman Company, New York.

ish gray tone of color; this brownish reflex from the lens is very distinct, especially when the pupil has been dilated. Hence in many cases the yellow color can be detected more easily in an old lens. The centre of the nucleus is stained more distinctly.

The dark lines of the anterior stellate rays also come clearly into view by focal illumination. Recapitulating once more the four characteristics that differentiate the old from the young lens we find:

That the senile lens is larger and more voluminous, as indicated by the fact that in age the anterior chamber is shallower than in youth.

That the senile lens is more yellowish, as shown by the brownish gray reflex.

That the senile lens has an altogether different consistence. If we hand this old patient a book to read he energetically refuses. The increased density of the lens, the enlargement of its nucleus and the loss of elasticity common to old age no longer permit the accommodative change of its form. These differences between the normal young and the normal old lens should remain thoroughly impressed on the mind.

In the dark room I will demonstrate an interesting phenomenon by means of a suitable small light, a delicate osmium incandescent lamp. Formerly it was thought that only one lenticular image was formed in the anterior portion of the lens of the normal eye, the anterior *Purkinje's* image. But *Hess* has shown that after the age of twenty-five years, a second upright image may be made to appear in the anterior portion of the clear lens. Its luminosity increases with age, and in old people it is nearly as bright as the anterior lenticular reflex. If the little lamp used by *Hess* is held laterally, so that its light enters the eye of an old patient, and the lamp is moved up and down, it will be perceived that, together with the corneal reflex, are two others that move up and down in the same direction as the light. The first of these is the anterior "reflex of the cortex"; its excursions are greater than those of the second, the "reflex of the nucleus." If the lens reflex seems at first to be single, it may be separated into two by moving the light about. It can then be seen that the reflex from the anterior cortex is somewhat more luminous than that from the nucleus. In elderly persons it is also demonstrable that the inverted posterior lenticular reflex is composed of a reflex from the posterior cortex and the posterior nucleus.

II

Diseases of the Conjunctiva and Cornea

5. Guiding Principle in the Analysis and Diagnosis of the Commoner Opacities of the Cornea

We are now in a position to study clinically the morbid conditions of the conjunctiva and cornea, but before we enter upon their individual diseases there is one subject with which I think we should be familiar, the clinical pictures presented by the commoner opacities of the cornea. We continually meet with opacities of the cornea, the nature of which is of fundamental importance in the diagnosis of the disease in question, whether it may be a keratitis secondary to a conjunctival disease, or a primary affection. For the disease in question, whether of the conjunctiva or of the cornea, may be diagnosed from the sort of corneal opacity in a great many cases. There is a certain clinical concept with which we have to deal and which should be thoroughly understood before we begin the study of the individual diseases of the outer segment of the eyeball, because we can then go forward so much more rapidly. Every academic teacher knows that it is difficult for students to find their way unaided among the corneal opacities. In spite of its diminutiveness the cornea presents a variegated mixture of clinical pictures. In addition to this, each individual morbid type presents variations. There is scarcely a diseased cornea that resembles exactly another suffering from the same type of disease, and it is not surprising that, lacking the necessary experience, students lose their way in this labyrinth of clinical pictures. I have drawn up this simple table in the hope that it may be useful as a guide; the commonest corneal opacities may be analyzed through the four questions it suggests. It hardly needs to be said that diseases do not really have such rigid outlines and that all clinical pictures are not included in the chart. The principle is to teach the student to investigate independently; if this aim is attained I shall be satisfied.

The multitude of corneal opacities is divided into two groups, fresh and old, for opacity of the cornea is not only met with in acute disease, but, unfortunately, is often permanent. The border line separating the fresh from the old, or very old, opacities cannot be sharply drawn, for there are many transition stages. The extremes may be learned, and then the student will gradually become acquainted with the transition forms. For the present it will suffice to answer the sharply defined interrogation: Is this a fresh, or an old process? In regard to this we have a certain infallible sign, which leads us to the consideration of two clinical concepts, the conjunctival and the pericorneal injection, with which we shall have to deal continually. Whenever there is an inflammation in the anterior segment of the eyeball, we meet with a hyperæmia as an accompanying, or local, phenom-

Analysis of Corneal Opacities:

- **I. Fresh Process (Ciliary Injection)**
  - **II. Loss of Substance (Ulcer)**
    - Ulcus eczematosum
    - Ulcus serpens
    - Ulcus catarrhale
    - Diplobacillus ulcer
    - Trachomatous ulcer
  - **II. No Loss of Substance**
    - **III. Superficial Opacity**
      - **IV. No Vessels**
        - Infiltrate
      - **IV. Vessels**
        - Pannus eczematosus
        - Pannus trachomatosus
    - **III. Deep Opacity**
      - **IV. No Vessels**
        - Keratitis interstitialis incipiens
      - **IV. Vessels**
        - Keratitis interstitialis provecta
        - Sclerotizing Keratitis
- **I. Old Process (No Injection)**
  - **II. Loss of Substance**
    - Ulcer in regeneration
    - Depressions, Facets
  - **II. No Loss of Substance**
    - **III. Superficial**
      - **IV. No Vessels**
        - Nubecula
        - Macula
        - Small Leucoma
      - **IV. Vessels**
        - Old pannus eczematosus or trachomatosus
    - **III. Deep**
      - **IV. No Vessels**
        - Interstitial Keratitis that has run its course
        - Sclerotizing Keratitis
      - **IV. Vessels**

enon in two different vascular regions of the eye, the regions of the conjunctival and of the anterior ciliary vessels. Hyperæmia of the superficial vessels of the conjunctiva scleræ we call conjunctival injection; that of the more deeply situated ciliary veins we denominate ciliary, or pericorneal, injection. In many violent inflammations of the anterior segment of the eye we find both of these vascular regions hyperæmic and injected, for there are numerous anastomoses between them. These two injections must be most accurately known. Even when they occur together they may be readily distinguished.

In our first patient the conjunctiva scleræ is reddened. The redness consists of a network of larger and smaller bloodvessels about the cornea, the individual vessels of which are clearly defined. That they belong to the conjunctiva is shown by the fact that they can be slid to and fro with the conjunctiva by pressure with the margin of the lid, and their superficial position is also indicated by their bright red color.

The conditions are quite different in the next patient, about whose cornea may be seen a narrow zone of markedly violet color, in which the individual vessels cannot be distinctly seen. The redness is diffuse, and the color is different from that in the other case. These indications show that the vessels occupy a deeper situation, that they lie in the layers of the scleral lamellæ. This may be proved by the fact that these vessels do not move with the conjunctiva when it is shoved to and fro. Somewhat farther from the cornea the veiled appearance of the vessels indicates their deeper location.

Pure types of these two forms of injection are met with in comparatively few cases; more frequently both vascular systems are more or less injected, because of the numerous anastomoses, but usually one or the other predominates, so that the condition is easily recognized.

Now we will return to the query why so much emphasis is given the presence or absence of a pericorneal injection when we see an opacity of the cornea. Clinical experience has taught us that ciliary injection is preeminently an accompaniment of diseases of the cornea, iris and ciliary body; hence, if we find a ciliary injection of the eye in which there is an opacity of the cornea, it proves that there is present either an acute keratitis, or a keratitis that is not yet well. Therefore, to answer the first question, whether the process is fresh or old, we have only to note the condition of the eye as regards injection.

I. Clinical Use of the Chart

Study of Progressive Ulcus Corneæ

Here is a patient whose eye is plainly much irritated. He blinks constantly, the eye lacrymates, he complains of pain. The irritated condition, which may be seen from a distance, demonstrates that this is not a case in which the disease has run its course. When the eye is open we may see by daylight an opacity in the centre of the cornea. The first question is: Fresh or old? There is a marked conjunctival injection over the entire conjunctiva scleræ, and in addition a very distinct dull violet pericorneal injection. Hence the process is fresh. With this differentiation one-half of the corneal opacities in the chart are excluded, and we pass at once to the second question: Is the opacity due to an ulcerated loss of substance in the cornea or not? Is an ulcer present or not? This question was chosen as the second because it is always the most easily answered. In order to determine whether a loss of substance is present it is necessary only to look into the eye from the side, or to use focal illumination, and we usually find that at one point on the surface we are able to see into an excavation of the parenchyma. The most marked phenomenon in our patient is a yellowish gray opacity in the centre of the cornea which is fresh, because the eye has both a conjunctival and a ciliary injection, and a distinct loss of substance in the cornea can be seen. This is the first ulcer of the cornea that we have clinically diagnosed.

An ulcer of the cornea is a loss of substance that extends from its surface into the parenchyma to a varying depth, and is caused by necrosis of the affected portion of the corneal tissue.

These circumscribed necroses of the surface of the cornea are produced in two ways. The surface may be sloughed by the action of acids, or alkalies, as in lime burn. leaving ulcers, but by far the most common cause is an infection. In practice we have most often to deal with infectious ulcers. Inflammatory agents enter the tissue from the surface, settle, multiply and excite inflammation by their toxic products. A preliminary condition to the production of most ulcers of the cornea is a lesion of the epithelium. The cardinal symptoms are inflammation, degeneration and regeneration. Increased numbers of cells appear in the vicinity of such settlements of the infectious agents; leucocytes wander into the cornea and form dense infiltrates of round cells between the corneal lamellæ in the affected portion. In addition to these pus cells, to the cellular exudate, we find anatomically signs of an œdematous saturation, and of excretion of fibrin in the tissues. If this exudation, together with the toxic action of the bacterial products of inflammation, is so great that the nutrition of the tissue is interfered with, the corneal lamellæ assume a finely granular appearance, swell and finally break down. When the necrotic portion is thrown off an ulcer remains.

During the course of an ulcer of the cornea, certain phenomena regularly occur which demand our attention. The question whether it is healing, or spreading, is of special interest. Every corneal ulcer has a progressive and a retrogressive stage. In this patient the prominent symptoms of irritation, together with its duration as given in his history, indicate that we are dealing with an ulcer in its first stage of development, but there are points in the clinical appearance of the ulcer itself from which we can determine whether it is spreading or not.

When a corneal ulcer shows a tendency to progress, the surrounding parts of the cornea are greatly infiltrated. This may be recognized from the fact that the base of the ulcer is gray, opaque and uneven, while its margins still appear yellowish and purulent, or very gray. The ulcer is also frequently surrounded throughout a large portion of its extent by a gray areola, or fine gray striæ extend from it in various directions. These are infiltrations that gradually fade into the surrounding cornea. In this way new areas in the neighborhood are invaded by the infiltration and break down into pus. Such an ulcer is spreading, is progressive.

In progressive ulcers the inflammatory irritation is not confined to the cornea alone. The iris is apt to be involved in the form of hyperæmia and inflammation. This distant action on the vessels of the iris will be taken up more in detail in the consideration of the various types of corneal ulcer.

The next thing we need to know is in what direction such an ulcer will progress, and in regard to this the clinical examination furnishes certain data. Two things are possible: the ulcer may progress in the direction of depth, or along the surface. In the former case it destroys one layer of the cornea after another and may finally result in perforation, a complication that leads to further trouble. In the latter case the extension is superficial. In both cases the progression takes place where the cornea is most markedly gray, or yellowish, where the tissue is most infiltrated with pus-cells. This condition is most marked at one part of the margin in our case. There are many cases in which the progress is simultaneously both superficial and deep. Having thus recognized an opacity of the cornea to be a fresh ulcer by means of this systematic interrogation and the clinical data, the next question concerns its origin. In practice we have three different causes of an ulcer of the cornea: 1. Ulceration of the cornea may occur in all forms of conjunctivitis, catarrhal, croupous and purulent. The details of the appearance and course of such ulcers will be given with the demonstration of those diseases. 2. The ulcers met with in keratoconjunctivitis eczematosa must be looked upon as a special form, which demands particular attention. 3. Ulcers of the cornea develop very often after slight injuries of the eye. We meet with them chiefly in adults of the laboring classes. This type is represented by the so-called ulcus serpens, with which we shall deal at considerable length, as it is of great importance to the physician.

The most important points in the differential diagnosis between these ulcers are as follows:

Ulcus serpens: 1. This disease attacks only middle aged or elderly persons. 2. It is usually situated in the centre of the cornea. 3. It spreads superficially. 4. It is usually associated with a severe iritis and an accumulation of pus in the anterior chamber.

Ulcus eczematosum: 1. This is a disease of youth, met with in children who are suffering from keratoconjunctivitis eczematosa. 2. It may appear anywhere on the cornea. 3. Its form is roundish. 4. In contrast to ulcus serpens it tends to progress deep into the parenchyma of the cornea, and, therefore, is very apt to result in perforation.

Ulcus catarrhale: 1. This form of ulcer is found only at the margin of the cornea. 2. It is usually of a sickle-shaped, or crescentic form. 3. It is associated with catarrhal inflammations of the entire mucous membrane which have a purulent secretion.

II. Study of the Corneal Infiltration

Our next patient is a child who has, near the margin of the cornea, a roundish gray opacity about as large as the head of a pin.

First question: Is it fresh or old? Answer: Fresh, for symptoms of irritation are present, consisting of photophobia and blepharospasm, and there is a distinct ciliary injection. Second question: Is there an ulcer present? Answer: No, for no loss of substance can be found. The opacity is a little focus over which the epithelium of the cornea is still present. Closer inspection shows that the epithelium looks delicately gray, and somewhat as if it had been pricked with needles. Our next question is: Is the opacity superficial or deep? This is the most difficult to answer of all the questions in this analysis. It requires a certain amount of experience to distinguish between superficial and deep lesions, for the cornea is only about a millimetre thick. The differentiation is made through the fact that superficial opacities of the cornea are always distinct, while the deeper ones are more or less veiled. In order to answer more easily this important, and frequently decisive, question of diagnosis, I advise that it be combined with our final question: Is it provided with bloodvessels, or not? The normal cornea contains no bloodvessels, but many corneal diseases and, likewise, opacities are associated with an active new formation of vessels, which are easily to be seen. This vascularization of corneal opacities is often so characteristic, according as it is situated in the superficial or deep layers, that it gives immediate information concerning the position and depth of the opacity itself. In the present case no bloodvessels are to be seen, the corneal opacity is recent, it is superficial, as is plainly evident from the elevation of the epithelium; hence, according to our guide, it can be an infiltrate only.

An infiltrate is nothing else than the first stage of an ulcer of the cornea. We understand an infiltrate to be an exudate composed of cells into the parenchyma of the cornea, a localized permeation of the tissue beneath the epithelium with lymphocytes or leucocytes; therefore the beginning of a circumscribed inflammation of the cornea. The epithelium over such an infiltrate suffers very easily and quickly, it loses its smoothness and may become somewhat elevated. The roundish form of the infiltrate is caused by this localized collection of cells. A concept of the little focus of inflammation presented by this patient may be obtained from a study of the microscopic preparations and drawings.

If the infiltrate becomes somewhat greater, i.e., if the exudation increases, *Bowman's* membrane may become implicated, the epithelium exfoliates still more, the infiltrated parenchyma becomes necrotic, and an ulcer of the cornea is formed.

An infiltrate may, however, be entirely resorbed without destruction of the affected portion of the cornea. Restitutio ad integrum begins, and the portion of the cornea hitherto opaque becomes clear again, but a result so fortunate is observed only in small and benign infiltrates, as a rule. When the infiltrate is deeply seated, we may become clinically aware of the destruction of corneal tissue only by noticing the resultant opacity and cicatrization.

Thus our ideas concerning the beginning of corneal ulcers have been perfected, and at the same time we have analyzed, with the aid of our chart, a new form of corneal opacity.

Among the superficial diseases of the cornea is the opacity caused by the so-called keratitis disciformis, a comparatively rare disease. The focus lies in the centre of the cornea, develops an infiltration beneath the epithelium which spreads like a disc in all directions, involving the deeper layers. The disease is, moreover, so tedious that its long duration is a point in its diagnosis.

III. Purpose of the Chart

Study of the Final Results of Corneal Ulceration

Our next patient has an opacity of the cornea that appears slightly gray on ordinary inspection. No signs of irritation and no ciliary injection are to be seen, so the process is evidently old. We also see, particularly with focal illumination, a loss of substance in the cornea, which proves the presence of an ulcer, although its syndrome is very different from those we have seen. The reason for this is that whenever an ulcer of the cornea, of whatever origin, comes to a standstill, all the necrotic tissue is cast off and the exudates in the margins, base and vicinity of the ulcer are gradually resorbed. We recognize this healing of the ulcer, clinically, from the fact that its base becomes gradually cleared up and its margins smooth. Active proliferation takes place in the epithelium of the cornea, which grows over the base of the ulcer until the defect is covered. Then we see, as in this patient, a loss of substance still, but it is no longer gray, it is covered by glistening epithelium and can no longer be stained by fluorescin. A little later and we shall scarcely be able to speak of a distinct opacity at all, because everything appears transparent, but still later, when cicatrization has set in, an opacity appears again on the site of the former ulcer. The corneal tissue displays a wonderful tendency to replace and fill out the defects produced by ulcers to the normal level of the surface, the restitution of the loss of substance taking place beneath the protective covering of epithelium.

The replacement of tissue in such a defect frequently does not equal our expectations: with no signs of irritation a distinct depression is left, covered with glistening epithelium. Such a depression is called a facet. As a rule, however, the defect is completely replaced by tissue. Two different processes contribute to this end.

In very small ulcers the proliferation of the fixed cells produces new corneal tissue, which assumes a normal arrangement and is capable of a high degree of transparency. The younger the patient the better is this regeneration of tissue.

In the overwhelming majority of cases the new tissue is of a different character, and healing takes place by cicatrization through a new formation of connective tissue. This newly formed connective tissue is constructed of gross fibres and is not transparent like the normal cornea, but forms a scar called leucoma, because of its gray white, or white appearance. In these processes, in which regeneration of the corneal tissue takes place along with a development of connective tissue, it is noteworthy that a *Bowman's* membrane once destroyed is never reformed, while *Descemet's* membrane may grow again.

Because corneal ulcers heal with cicatrization we are able for years, often for a lifetime, to read in a patient's eye that he has suffered from one or more ulcers by noting the corresponding opacities present. In the patient now before us the eye is perfectly free from irritation, the disease ran its course long ago. The opacities are superficial and contain no vessels. The epithelium over them is perfectly smooth and lustrous. Accordingly we diagnosticate, guided by our chart: Old opacities of the cornea, maculæ. If they are very delicate and translucent, we call them nubeculæ. All of these opacities have the same origin—they are connective-tissue scars, left after the healing of ulcers of the cornea.

This is still more distinct in our next case. Healing took place long ago, there is no longer any loss of substance, the corneal opacity is superficial, intensely white, with a tendinous lustre. This is a leucoma corneæ due to previous ulceration, and its glistening white appearance is caused by the fact that this layer of connective tissue is rather thick, an indication that the preexisting ulcer must have penetrated deeply into the parenchyma of the cornea. When such ulcers are of considerable extent healing may take place with the aid of a marked neoplastic formation of vessels in the cornea. Hence, we are apt to find a greater or less number of bloodvessels in extensive leucomata.

6. Analysis of Vascularized Opacities of the Cornea

Although the ophthalmological text-books consider separately the morbid syndromes of the conjunctiva and cornea, such a division cannot be followed in practical study. Emphasis has been laid a number of times upon the fact that there is quite a number of diseases of the conjunctiva in which the cornea may become sympathetically involved. There is scarcely a part of the human body in which, on so limited a territory, such a multiplicity of morbid syndromes may be found as in the cornea. Of the thousands of clinical pictures to be seen in the cornea, no one is exactly like another; a law of pathology which, it is granted, is fundamentally true to a greater or less degree of all organs of the body, but is rarely displayed in so striking a manner elsewhere. The most notable property of the cornea, clinically, is its transparency; this property must suffer change from the least morbid alteration in its tissue. In fact, most diseases of the cornea produce opacity, and it is from the nature and condition of these opacities that we are able to recognize the disease that is present. Whoever investigates this subject will find that the opacities in the diseased cornea have been particularly noted and described.

We have begun the systematic analysis of the most important corneal opacities commonly met with in practice, but have considered only a small portion of them. To-day we will study the *vascularized* opacities. These are very common, as is shown by the chart: on the one side, pannus eczematosus, pannus trachomatosus, pannus regenerativus, the vascular fasciculus, vascular leucomas; on the other the morbid processes that appertain to interstitial or parenchymatous keratitis.

A.—Superficial Vascularized Opacities

Our first two cases present unmistakable signs of irritation; photophobia, pericorneal and conjunctival injection. The most striking symptom is the presence of bloodvessels in the distinctly visible opacity of the cornea. As a matter of fact there are two forms of vascularization, superficial and deep.

1. Acute Pannus

When there is a superficial vascularization of the cornea, bloodvessels may be seen running from those of the conjunctiva into the cornea. They are sharply defined and clear, are of a vivid red color, have a very characteristic manner of branching which resembles the ramification of a tree, and the surface of the cornea is usually somewhat uneven. A sort of granulation tissue pushes its way into the cornea. The epithelium is elevated irregularly by the superficial vascularization, so that irregular elevations and depressions are to be seen, as on the surface of a granulating wound. The name of such a superficial vascularization is given by our guide as pannus.

Pannus is a vascular granulation tissue which grows into the cornea from the conjunctiva between the epithelium and *Bowman's* membrane, partly beneath the latter. Numerous little infiltrates are formed at the margin of the cornea, and between these ramify numerous vascular twigs from the superficial marginal network. Although the student may see a great many of these cases, it is always difficult to obtain a plastic conception of these processes in the cornea. That he may learn and know them an explanation of these slides and drawings may be useful.

We see that the deeper lamellæ of the cornea are perfectly normal; that the pannus is therefore a superficial affection. This superficial disease consists of a proliferation of numerous neoplastic bloodvessels from the conjunctiva between the epithelium and *Bowman's* membrane, accompanied by young connective tissue in which lie larger and smaller groups of round cells in many places. *Bowman's* membrane may be destroyed in places by this granulation tissue. In grave cases the entire surface of the cornea may be occupied by such a newly formed vascular connective tissue, rich in nuclei. Turning to our patient again, we see that the pannus is a disease of the conjunctival layer of the cornea, and hence we must inquire in every case by what conjunctival disease it is caused.

This clinical concept leads immediately to a new association of ideas, for we meet with pannus in practice, principally, in two diseases of the conjunctiva, keratoconjunctivitis eczematosa and conjunctivitis trachomatosa, in which it is known as pannus eczematosus and pannus trachomatosus. The diagnosis pannus is insufficient, we need to know whether we have to deal with a trachoma, or an eczematous keratitis.

The most important characteristics for the differential diagnosis between pannus eczematosus and trachomatosus are as follows: Keratitis eczematosa is a disease of childhood; therefore, eczematous pannus is met with chiefly in young persons, while trachoma patients are usually middle aged, or elderly. Eczematous pannus may also start from any place on the cornea, while trachomatous pannus almost always begins at its upper margin. It is also characteristic of the latter that it is terminated for a long time by a sharply defined horizontal line. It is only in the latter part of the course of grave cases that pannus trachomatosus spreads over the entire cornea, but even in such cases the upper portion is more seriously affected. Such a condition will naturally lead us to look immediately for other trachomatous changes in the conjunctiva. Thus, the aspect of such a superficial vascularization may give important information concerning the nature of the disease present and enable us to detect the fundamental trouble.

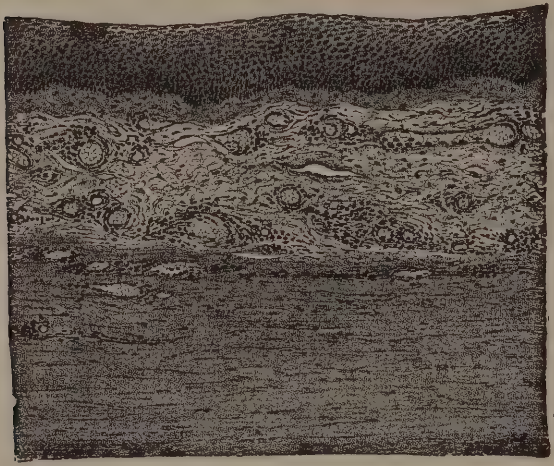


FIG. 10.—Pannus.

2. *A Case of Old Pannus*

Vascular opacities of the cornea have their fresh and their old stages, and it is characteristic of the human eye that corneal opacities are capable of an extensive retrogression. In this the vessels take part. They persist longest in the processes of retrogression, as a rule, and then we can recognize again from their position and manner of extension that this or that disease has been present in the eye. This patient furnishes an example.

There are no longer any signs of irritation, the disease has run it course, and yet some ramifying vessels may be perceived in the opacity and traced to the conjunctiva, proving them to be superficial. Our diagnosis is an old pannus. As regards the involution of these vessels it has been believed that they become largely obliterated, and there is no doubt that the superficial vessels in the cornea are capable of an extensive involution, but isolated vessels can be found by careful examination even in the oldest superficial opacities of the cornea. On the other hand, in our treatment of pannus, e.g., in jequiritol ophthalmia, frequently also when all phenomena of irritation are absent, such an extensive vascularization comes into view in so short a time that we are forced to admit the persistence of vessels, even when they had been looked for in vain.

3. *A Case of Pannus Regenerativus*

In this patient a little ulcer may be seen near the margin of the cornea, to which little vessels extend from the limbus. Clinically, this is a good sign, such vessels serve for regeneration, and this form of vascular opacity is called pannus regenerativus. This can hardly be mistaken for ordinary pannus, because the latter is more diffuse and ulcers do not appertain to its clinical picture.

4. Opacity with Vascularization: Keratitis Fascicularis

In this case again vessels may be seen in the cornea, but the opacity in which these little vessels run is not to be mistaken for a pannus. It is narrow and tonguelike, whereas the vascularized opacities we have been studying, especially pannus, involve a greater area of the cornea. This is the so-called keratitis fascicularis, which will be considered in detail presently. It is sufficient just now to note the superficial position of the vessels and its characteristic form.

5. Species of Vascularized Opacities: Vascular Leucoma

This is still another case of vascular, superficial opacity. It is sufficiently distinct in its whitish, glistening tissue, to be recognized at a distance as a leucoma. Numerous fine and coarse vessels, a large part traceable to the margin of the cornea, may be seen in this cicatricial tissue. The leucoma, which occupies a large portion of the cornea, resulted from a grave ulcer that formerly existed at this place, and this case illustrates the fact that the healing and cicatrization of large defects in the cornea takes place in connection with a considerable new formation of vessels. The tendonlike appearance of such opacities indicates the diagnosis at once; even on the street persons with such leucomas may often be recognized from afar.

B.—Deep Vascular Opacities of the Cornea: Keratitis Parenchymatosa

The picture presented by the next patient is altogether different. The cornea has a red color, which can come only from bloodvessels, but it is a dirty gray-red, because the vessels are obscured by the overlying opaque layers of the cornea, so that it is difficult to discern individual vessels with the naked eye, except at the margin of the cornea, where the task is rather more easy. At that point, too, the vessels seem to suddenly end, contrasting in this respect with those of the superficial vascularization. They disappear behind the limbus because they plunge into, or more correctly emerge from, the depth of the sclera. Finally, it can be perceived at the margin of the cornea that they consist of individual, rather parallel vessels. Hence we speak of a broomlike branching of the vessels in the deep vascularization.

Even when a keratitis parenchymatosa has completely run its course, throughout the life of the patient indeed, a part of these vessels remain in the cornea, so that these persistent, deep corneal vessels can be utilized in the analysis of a case of this disease even in it oldest stages.

A phenomenon so peculiar and so important clinically as a new formation of bloodvessels in the otherwise non-vascular cornea cannot be passed by without at least one query as to its physiological significance, though the sum of our knowledge in this matter is still very slight. It seems to be incorrect to say, with *Fuchs*, that the superficial vessels serve for purposes of repair, while the deeper ones of interstitial keratitis pertain to the clinical picture of inflammation. We must admit in all cases that the development of bloodvessels in the cornea indicates a reaction of the tissue to irritants that may be of mechanical or chemical nature, but usually consist of abnormal products of metabolism which stimulate the growth of the vessels. The functions of these vessels therefore differ according to the stimuli.

They may perhaps furnish certain protective substances to the inflammatory foci that cannot be supplied by the lymphatics alone, and, in grave ulcers, they undoubtedly serve to supply material for the filling up of the defects. Above all they are of importance in the clearing up of corneal opacities. This is shown by the fact that in the involution of such opacities the first places to clear up are in the immediate vicinity of the vessels.

I advise that this chart be firmly fixed in the mind. With the four questions: Fresh or old process? Ulcer or no ulcer? Superficial or deep? Vessels or no vessels?—we can deal quickly with the great majority of corneal opacities. It may be confusing for a time, but by this method the power for independent investigation will be acquired. Given a great number of patients with various corneal troubles, the student with a working knowledge of this chart will be able, independently, to analyze them into separate groups and to diagnose with certainty.

A demonstration of how much more can be seen with the *Zeiss*' corneal microscope will be of advantage. We will use the objective a_2, and the ocular 2, thus obtaining an enlargement of 24.

There remain two important points for us to consider in the analysis of vascularized opacities of the cornea. First, it can be seen in this patient that the movement of the blood may be directly seen in the newly formed superficial vessels. We are able to watch this movement chiefly in the veins, for it is evidently centrifugal, from the centre toward the periphery of the cornea. This is of great clinical and diagnostic interest, for we cannot see this movement of the blood in corneal diseases as long as the inflammation is at its acme, but only after the inflammation has abated and the current has become slower. We can therefore recognize the abatement of the inflammation from the appearance of a visible movement of the blood in the newly formed corneal vessels. Another phenomenon which is of interest is that in many cases we can verify a dependence of the movements of the blood column upon the pulse-beat.

A second reason why vascularized corneal opacities should always be examined with *Zeiss*' corneal microscope is this: The conditions of corneal vascularization are, actually, more complex than is indicated in our schematic interrogatory. It is only for didactic reasons, not to make the subject too complex at first, that we have said that the distinctly visible, superficial, ramifying vessels in the cornea pertain to superficial, conjunctival inflammations, while the broomlike vessels indicate a disease of the deeper layers. It is certainly correct that the subepithelial vessels coming from the superficial marginal network usually exhibit a treelike branching, while those from the deep marginal network as a rule follow a direct course. The explanation of this peculiar arrangement is that the superficial vessels between the epithelium and *Bowman's* membrane can develop to a certain degree unhindered, following no prescribed routes. The deeper vessels, on the contrary, run between the lamellæ of the cornea, which force them to follow more direct courses. It may also be seen that the course of the parent stem of the arboreally branching, superficial vessels is considerably different from that of the venous channel, while the arteries and veins of the deep vessels in the lamellæ lie close together and parallel. It may also happen that vessels belonging together are at different depths, and we meet with an anastomosis between vessels at different depths, especially in cases of grave cicatrization in the cornea, when an oblique course of the vessels is not a rare occurrence.

The subepithelial vessels develop in such a manner that individual sprouts push forward into the cornea, while the beginning of the development of the lamellar vessels is always by means of a loop representing a double vessel. Although we are able to thus easily classify anatomically the two kinds of vascularization, yet it is maintained clinically that the lamellar vessels are not characteristic of keratitis parenchymatosa and are by no means confined to that disease.

According to the researches of *Augstein* and of *Brueckner*, which every clinician who uses *Zeiss*' loupe regularly can confirm, there is no doubt that we regularly meet with the development of lamellar vessels in other corneal diseases. If this development is traced more accurately the lamellar vessels may be subdivided into three groups:

Lamellar vessels in the superficial, rarely in the middle, layers of the cornea. These are met with chiefly in keratitis eczematosa, especially when the ulcers are near the margin. Not infrequently it can be observed that these vessels divide into tufts. Among other corneal affections that may result in the development of individual, superficial lamellar vessels may be mentioned encapsulated foreign bodies and mild cases of lime burn.

We shall now concentrate our attention on patients with keratitis parenchymatosa, of which it is characteristic that the vessels run chiefly in the middle layers of the cornea. A little practice will enable the student to demonstrate this with the aid of the binocular vision afforded by this invaluable instrument. In some cases superficial lamellar vessels are also present.

Finally, we can differentiate lamellar vessels in the deepest layers of the cornea. They are met with, together with the superficial, during the healing of large ulcers. Such deep vessels are especially frequent in the scars left by ulcus serpens. It is of diagnostic value to know that vessels may also appear in the deepest layers of the cornea in cases of chronic iridocyclitis. Even more striking is the fact that the lower margin of the cornea becomes a site of predilection for the development of such vessels in iritis. Whether this has any connection with the localization of *Descemet's* deposits is questionable. Complicated as these conditions seem at first glance, the student will soon learn to interpret correctly the clinical pictures on the cornea if he will first establish whether he has to deal with the superficial, subepithelial, arboreally branching vessels, or with the lamellar vessels which disappear at the margin of the cornea and follow a more direct course.

7. Clinical Syndrome of Acute Catarrhal Conjunctivitis and Its Etiologic Diagnosis

Our patient tells us that he has had a violent inflammation of both eyes for the past couple of days. One eye was attacked first, and soon afterward the other. He complains somewhat of photophobia, and of a feeling of heat and burning in the eyes. One eye blinks constantly and feels as if a foreign body was lodged in the conjunctival sac. He says also that his trouble is greatest in the evening, especially in artificial light. There may be also disturbances of vision that can be relieved temporarily by rubbing the eyes, but soon return. On awakening in the morning his lids are so glued together that he has difficulty in opening them.

The clinical picture is very characteristic. The lids are slightly swollen, reddened, with here and there dried secretion, especially among the lashes. The greater part of this secretion has come from the conjunctival sac and become dried during the night, but the secretion from the *Meibomian* glands may also be increased in these cases.

Larger quantities of fresh, muco-purulent secretion have accumulated at the outer, and still more at the inner canthus.

If the lower lid is drawn down from the eyeball a still further quantity of more dilute secretion is to be seen, flecked with bits of mucus floating about in the conjunctival sac. The conjunctiva palpebræ is much reddened and swollen, though smooth and glistening. The swelling is recognized from the fact that the *Meibomian* glands no longer shimmer distinctly through the mucous membrane. The lower transition fold bulges forward in thick rolls. The inflammation also involves the conjunctiva bulbi, which is very much reddened, almost bluish. The redness is most marked toward the transition fold. The condition might almost be called chemosis.

At the inner canthus we find the plica semilunaris and caruncle involved. If we examine the upper portion of the conjunctiva bulbi we find here also a fresh redness, as well as a number of punctate and flecklike red spots, which are subconjunctival hemorrhages, such as we are accustomed to meet with in the upper part of the conjunctiva bulbi in these diseases. Finally, the same redness and swelling of the mucous membrane is to be seen in the upper transition fold.

The visual troubles met with when the secretion is so great can be readily understood. If a bit of mucus happens to lie on the cornea it will disturb the refraction of the light, and variegated circles may be seen around the flame of a lamp.

In this patient the disease is fresh, the mucous membrane exhibits redness, swelling and abnormal secretion, the secretion has a mucus character. Clinically, therefore, we are dealing with a conjunctivitis acuta catarrhalis.

We still lack the etiologic diagnosis. It will be recalled that all conjunctival inflammations are divided into two groups: those due to purely chemico-physical agents and those caused by infection. As the history in this case gives no suggestion of the first cause, the probability is that we have to deal with an infectious disease of the eye. This can be ascertained with certainty only by a bacteriological examination of the diseased conjunctiva and of its secretion.

In order to understand the etiology of conjunctival inflammations, the student needs to be acquainted with three things:

First, the method of examining bacteriologically the conjunctiva and its secretion.

Second, the germs contained in the normal conjunctival sac; for if the physician is to consider the bacteriologic content of a secretion to be pathologic, especially if he is to hold the germs found to be the agents productive of the inflammation present, he must know how the mucous membrane behaves with regard to its bacterial content under normal conditions.

Finally, it is well for the physician to learn what pathogenic agents are possible factors in acute catarrhal conjunctivitis, in order that he may know what prophylactic and therapeutic methods should be employed. I repeat that the etiology cannot be determined from the clinical picture of such a disease as the one we are now studying. We are unable to say from its appearance by what pathogenic germ an acute conjunctivitis is caused, because there are no absolutely typical clinical pictures characteristic of the different kinds of infection. It is true that the ophthalmologist has frequent opportunity to make snap diagnoses in regard to the causes of these inflammatory conditions, because the specialist interprets more easily the relatively characteristic signs of the different infections, but this does not alter the fact that we are often apt to err in such snap diagnoses, and that for both the accurate investigator and the physician the clinical diagnosis of such a conjunctivitis must be supplemented by the bacteriological findings.

A. Method of Examining Secretions from the Eye

The technique of this examination is familiar to all who have studied bacteriology. There are several rules to be observed in this examination of secretion, if the result is to correspond to our expectations and reveal the pathogenic agent.

We take some mucus, or pus, from the conjunctival sac by means of a platinum loop. We must not take lachrymal fluid only, for we would seek vainly therein for the morbific agents. The secretion must also be taken from the conjunctiva itself and not from the margin of the lid, or the canthus, for in these places the agents of the conjunctivitis become mingled too easily with the parasites of the skin, chiefly saprophytic staphylococci and xerosis bacilli.

Disappointment must not be felt when no bacteria are discovered in this manner. Everything depends on the examination being made at the proper time. As soon as the disease begins to abate the pathogenic agents disappear, often with remarkable celerity. The best time for an examination is during the rise of the disease, or at its acme. The secretion upon the loop may be examined microscopically at once, or cultures may be prepared from it. The busy ophthalmologist must frequently content himself with a stained slide, and the general practitioner cannot be expected to prepare cultures. The general practitioner, indeed, rarely makes a microscopical examination, because he has more important matters to attend to than the examination of conjunctival secretions. Nevertheless he should know how to make such an examination, for he may need to make use of the technique sometimes, as in a case of blennorrhœa.

The secretion is smeared as thin as possible on the slide, for the thinner the layer the better the stain is taken. The smear is then dried in the air and fixed as usual by passing it three times through a flame. It is then stained either with *Lœffler's* methylene blue, or with a very dilute solution of fuchsin. A second smear is then prepared and stained by *Gram*, by means of which we differentiate between the *Gram* negative and the *Gram* positive bacteria.

This differentiation is of great value in the diagnosis of infectious diseases of the conjunctiva, as will appear in the study of blennorrhœa. The smear fixed by passing it through the flame is stained with carbolgentian violet, laid for a short time in a solution of iodine and potassic iodide, decolorized in alcohol until no more stain comes off, rinsed off with water, and finally restained with an aqueous solution of saffranin. After it has been rinsed again with water it is dried and is ready for examination with the oil immersion. In alcohol, *Gram* negative bacteria lose the violet stain and are restained by the red dye of saffranin, while the *Gram* positive retain the blue violet color throughout the procedure.

If cultures of the pathogenic bacteria suspected or known to be present are required, the most various culture media should be employed, and both aërobic and anaërobic cultures prepared. The secretion is inoculated first on agar, then on *Lœffler's* serum and ascites agar, finally on blood agar. The advantage of this method is that, knowing, as we do, the conditions that govern the growth of the bacteria met with in practice in the eye, we are able to isolate the sensitive germs which are easily overwhelmed by others. For anaërobic cultures we place the tubes in a glass cylinder with an air-tight cover, the bottom of which is covered, according to the method of *Buchner*, with pyrogallic acid and dilute liquor sodæ. A clinic is not a suitable place for us to go into further details in regard to this subject.

B. The Bacteria of the Normal Conjunctiva

We must now consider a question that is not to be avoided. Even if we find microorganisms on the slide, this does not prove that they are the agents that produced the disease in our patient. The normal, uninflamed conjunctiva contains microorganisms. Normally, there are no microorganisms in the conjunctival sac of an infant immediately after birth, but after a short time they have lodged there. This is readily understood, for the conjunctival sac opens outward so that the greatest variety of microorganisms can very easily enter from the air, the water of the bath, the surface of the neighboring skin, the clothing, the fingers and other objects. Soon, however, the bacterial flora on the normal conjunctiva assumes a characteristic aspect, so that we find in children a collection quite similar to that met with in adults. Of course all kinds of microorganisms may be met with in the conjunctival sac, and whoever wishes to take the trouble may always find here germs that have not been met with before.

We also know that the germs contained in the normal conjunctiva depend on external and personal conditions. The dissimilar environment, the nature of the occupation, and the degree of the personal cleanliness have such an effect that we meet with a greater number of germs in persons of the laboring classes, who work in a dusty atmosphere, than in people who pay more attention to hygiene and the care of the body. In the morning also the number of germs on the conjunctiva is rather greater than in the evening, because the lids are closed during sleep. It has been demonstrated by *Schirmer* that with the cessation of winking the conduction of the bacteria toward the nose also ceases. In this respect we may consider the regular act of winking as a physiological self-cleansing of the eye. *Marthen*, *Bach*, *Bernheim* and *Morax* have also demonstrated that the germs of the conjunctiva augment considerably when the eye is bandaged, because both the movement of the lid is stopped and the temperature is elevated beneath the dressing so as to favor the development of microbes. Another fact that should not be overlooked is that differences in the bacterial content of the normal conjunctiva are met with, according to the methods of examination and the culture media employed. In spite of all these factors by which the bacterial condition of the normal conjunctiva may be influenced, we can note the following certainties.

It is positive that the normal human conjunctiva is not sterile. Its content of germs is always less than that of the neighboring margins of the lids. The bacteria regularly met with on the normal conjunctiva are:

1. Xerosis bacilli.
2. White staphylococci of little or no pathogenicity.

Rarely pneumococci and the yellow staphylococci are seen. Streptococci and influenza bacilli are extremely rarely to be met with on the normal conjunctiva. In addition to these there may be occasionally met with all manner of saprophytes and pathogenic germs such as cocci, sarcinæ, and hay bacilli. Frightful as this bacterial condition appears at the first glance, troublesome as the fact is that the surface of this wonderful organ is not sterile, yet, in harmony with clinical experience in the results of operations, virulent pus agents are not present on the normal conjunctiva in the majority of cases. They are met with in a small percentage of cases, in individual examples, which suffice to keep us vigilant, but on the whole the normal conjunctiva is comparatively inapt to harbor infection. The reason for this deserves explanation, for the moist surface of the conjunctival sac would seem to offer a desirable habitat to the various kinds of microorganisms which have abundant opportunity to enter. The temperature in the conjunctival sac is about 2% lower than in the mouth, because of the exposed position of the eye, the evaporation of the lachrymal fluid and the comparatively moderate amount of blood in the normal conjunctiva. The lachrymal fluid is not a good culture medium for bacteria. It contains 0.5% albumin, 1 to 1.5% NaCl, 0.1 to 0.2% Na_2Co_3, 0.05% $MgSO_4$, 0.05% Na_3PO_4. Every attempt to grow a number of bacteria in such a fluid demonstrates its inferiority to our ordinary culture media. The view has been advanced repeatedly that the tears have a bactericidal action, but too little distinction has been made as to whether the tears furnished an unsuitable medium for bacteria, or were in their effect directly bactericidal, or inhibitive of the virulence of the bacteria. The characteristic first mentioned can alone be ascribed to the tears; they form a bad nutritive medium for most kinds of bacteria. They have no directly bactericidal quality, for they lack those bodies of the serum, bacteriolysin and hemolysin, which are the agents of bactericide activity. Furthermore, the tears contain no agglutinin, and no antitoxin can be found in them, even when large quantities of diphtheria antitoxin have been injected into the bodies of the persons used for the experiment, as was proved by *Demaria* in disproof of statements to the contrary. On the other hand there are species of bacteria, especially of the diphtheria group, like the xerosis bacillus, which multiply in stagnant lachrymal fluid after extirpation of the lachrymal sac. The effect produced by the lachrymal fluid in the normal conjunctival sac is chiefly mechanical. The microbic population of the conjunctiva is constantly reduced by the current of lachrymal fluid toward the nose. Even the conjunctival mucus favors the growth of bacteria but little, according to *Bach.*

Saprophytes interfere with the growth of other germs by a sort of antagonism, and it has been supposed that the usual inhabitants of the normal conjunctiva, the white staphylococcus and the xerosis bacillus, hold in check, in some such manner, the growth of other pathogenic bacteria, but it has been proven that these bacteria do nothing of the kind. Indeed, we have certain reasons to think that the ordinary parasites of the conjunctiva favor the development and multiplication of many other agents. Thus the xerosis bacilli act as nurses to the pathogenic *Koch-Weeks'* bacilli, and promote their multiplication both in cultures and on the conjunctiva. *Koch-Weeks'* bacilli are cultivated with difficulty in the absence of xerosis bacilli. On the other hand, we see that the development of pathogenic bacteria in the conjunctival sac gives the saprophytes a better environment in which to live, so that xerosis bacilli from an inflamed conjunctiva can be cultivated with less difficulty and in greater luxuriance than those from a normal mucous membrane. Whether the saprophytes are able to change their character and become virulent has not yet been sufficiently investigated.

C. Etiology of Acute Catarrhal Inflammations of the Conjunctiva

There are two preeminently common forms of acute conjunctivitis of this nature: 1. *Pneumococcus conjunctivitis*, produced by infection with the *Fraenkel-Weichselbaum* diplococcus lanceolatus; 2. *Conjunctivitis due to the Koch-Weeks' bacillus.* The therapeutic and prophylactic measures we adopt depend on the differentiation; the spontaneous course depends on the etiology. Every student should study the cultures and slides until he has an intimate acquaintance with the etiology of acute catarrhal conjunctivitis, and should impress on his memory the characteristic clinical picture. It is also well for him to investigate his own conjunctiva bacteriologically, by way of both cultures and smears.

3. Influenzal Conjunctivitis

A third form of acute conjunctivitis is produced by the influenza bacillus. I am unable to exhibit a case, for it is comparatively infrequent and principally observed during an epidemic of influenza. It is a conjunctivitis of varying intensity that has been met with in both infants and adults.

4. *Staphylococcal Conjunctivitis*

Still another group of acute catarrhs of the conjunctiva is caused by the ordinary pus agents, the staphylococci. Staphylococci are among the ordinary inhabitants of the normal conjunctiva, and it should not be forgotten that attempts to excite a catarrh by inoculations of pure cultures of the staphylococcus pyogenes aureus upon the normal conjunctiva have failed, and yet I agree with those ophthalmologists who recognize the existence of a staphylococcal conjunctivitis, even though it is comparatively rare. It need not be taken into account in epidemic forms, but according to my observations it must be considered in the following forms of acute catarrhal conjunctivitis:

(a) *Infantile catarrhs* from which the gonococcus has been excluded.

(b) *Following extraction of cataract.* In this case of acute conjunctivitis in a patient operated on for cataract a few days ago no microorganisms are to be found in the secretion except xerosis bacilli and great numbers of staphylococci aurei. We can easily imagine that in such cases these pus agents, that have previously been indifferent, find a better medium as a result of the mechanical and chemical irritation produced by the operation and so accentuate the conjunctival irritation until the picture of an acute conjunctivitis is produced.

(c) *Irritations of the eye* with staphylococci. Such irritations of the eye may be produced experimentally by introducing dust into the conjunctival sac and inoculating the conjunctiva with staphylococci, when conjunctivitis will be produced. Such irritations with dust may be produced in ordinary life either accidentally or purposely. During labor the conjunctiva of the infant may become so sensitive as to furnish a favorable field to the pus agent.

(d) *Acute conjunctival catarrh following blepharitis and eczema with staphylococci.* It is not at all unusual for the chronic hyperæmia of the conjunctiva to burst forth suddenly into an acute catarrh in cases of blepharitis and impetiginous eczema. Here also we meet with staphylococci in such masses that it is impossible to doubt their etiologic significance in such exacerbations.

(e) *Croupous conjunctivitis with staphylococci.* Finally, staphylococci have been found by quite a number of authors in forms of conjunctivitis associated with the formation of membranes.

In very rare cases of acute conjunctivitis pneumobacilli, bacteria coli and streptococci act as agents, but as compared with pneumococci and *Koch-Weeks'* bacilli they are of little importance in this part of the world.*

8. Course and Treatment of Acute Catarrhal Conjunctivitis

We come now to the practical treatment of these diseases. If this treatment is to be proper we must first know their courses, for only he who knows the spontaneous course of a disease knows what to treat and what measures to use. Therefore I shall sketch briefly the ordinary course of these diseases.

The Course of Pneumococcal Conjunctivitis

The inflammation develops very rapidly and involves, as a rule, both eyes. The margins of the lids, especially of the upper ones, become rosy and œdematous, and the bulbar conjunctiva particularly becomes very red. Indistinct little hemorrhages appear in the upper part of the conjunctiva bulbi and soon acquire a yellowish red tint. The disease reaches its acme quickly, usually in one or two days. The striking redness of the conjunctiva bulbi is characteristic of this stage. At the same time there is a great discharge of thin, watery secretion which contains bits of fibrin floating in it.

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* In 1914 Vail described a purulent conjunctivitis characterized by ulcers of the conjunctiva, a tender, swollen preauricular gland, an extension of the inflammation to the lacrimal sac, and the formation of pustules between the eye and the ear, which he believed to be the first case on record of a squirrel plague conjunctivitis. Experiments on guinea pigs with scrapings from the ulcers induced a similar disease characterized by a bacillus believed to be incidental with one pronounced by McCoy and Chapin to be the agent of squirrel plague.—Ed.

There are, of course, mild and severe cases of this disease. In grave cases the discharge is quite purulent, or slight membranes may be formed on the transition folds. It is therefore essential, in a clinically croupous conjunctivitis, that we should not assume the presence of a diphtheritic, or streptococcal infection. As a rule, however, pneumococcal conjunctivitis is a benign affection which disappears nearly as rapidly as it comes. Indeed, it is characteristic of this disease that it ends in crisis, quite like a pneumococcal infection of the lungs. The pneumococci then disappear very quickly from the secretion and cannot be found in it shortly after the disease has reached its acme. The discharge then lessens rapidly and the redness of the mucous membrane fades, so that recovery is complete in from eight to ten days. The etiologic diagnosis may often be established from the purely clinical observation of the rapid attack, the critical subsidence and the characteristic redness.

The disease is not met with uniformly everywhere, for while it is common in Germany, England, Switzerland, Russia and the United States, it is rare in Egypt, that breeding-place of other acute ophthalmic inflammations. In individual countries, also, its occurrence is very irregular. In general it may be said to be more common in the northern lands and to incline to the colder months of the year.

That the pneumococcus is the etiologic factor in such an inflammation has been demonstrated by inoculations with the discharges from the eyes and with pure cultures. Pneumococcal conjunctivitis may appear epidemically, especially in schools, but there is, as in almost every disease, an individual predisposition. Children are more susceptible than adults, so much so that pneumococcal conjunctivitis may almost be termed a children's disease. In epidemics there are many individuals who remain immune to the infection, and in experimental inoculation success is obtained in only a fraction of the subjects. Sporadic cases, too, result in no more cases, even though abundant opportunity is afforded for transmission.

Infection doubtless takes place by contact, but as many persons have pneumococci on their healthy conjunctivæ, or have such germs in their mouths, it is quite possible for this conjunctivitis to originate from autoinfection, the microbes becoming more virulent, or the power of the organism less strong.

Although this conjunctivitis is benign, complications may occur. In the first place we meet with a very marked nasal catarrh in connection with this conjunctivitis, which may precede the conjunctival trouble as a cold, or may be caused by the secretion. In the eye itself marginal infiltrates and small ulcers occasionally appear, but generally we need not fear corneal complication in a pneumococcal conjunctivitis. Iritis is met with more frequently, even without involvement of the cornea, and may outlive the conjunctival disease. Its genesis must be attributed to a resorption of toxines. In rare cases the development of follicles has been noted, but usually these have previously existed.

The Course of Conjunctivitis Caused by the Koch-Weeks' Bacillus

The course of an acute catarrhal conjunctivitis caused by the *Koch-Weeks*' bacillus is similar. After a short stage of incubation the palpebral margins begin to swell, and the secretion of the *Meibomian* glands may be augmented. The discharge from the conjunctiva becomes extremely copious, while the membrane itself is much swollen, though smooth and glistening. The fluid, muco-purulent secretion glues the lids together in the morning and gathers in large quantities at the canthi. The conjunctiva bulbi is much reddened, and we find in its upper part the same hemorrhages as in pneumococcal conjunctivitis. In this infection mild cases are met with, but in general the symptoms are more violent than in that produced by pneumococci. The patients complain of very great photophobia as well as of severe pains about the orbit and in the superior maxilla. In such cases the preauricular gland is apt to be swollen. The syndrome may be that of a grave purulent conjunctivitis and remind one of blennorrhœa. The formation of membranes on the conjunctiva of the transition folds may be transiently met with.

The disease lasts on an average for two to four weeks, but with improper treatment a hypertrophy of the papillary bodies may persist for a longer period, especially in the upper transition fold.

Several complications are possible. A severe coryza is not rare. Little blebs, or even true efflorescences, may appear at the limbus. Marginal infiltrates are to be observed in the cornea itself, though serious ulcers are comparatively rare. They are to be feared in adults, rather than in children.

The infection is spread through the secretion. It needs to be borne in mind that, long after the acute symptoms have subsided, the secretion is able to spread the infection, and it should be emphasized that the infectious secretion from the conjunctiva may pass through the lachrymal passages into the nasal cavity and into the mouth, and from there be spread by drop infection in speaking or coughing. The *Koch-Weeks*' bacilli are very sensitive to drying, and when dry die very quickly; but as long as the secretion is moist it is able to infect human conjunctivæ without exception, as has been proved by experiment. I can subscribe the statement from my own experience that this type of conjunctivitis is one of the most contagious diseases known. On animals these bacilli are absolutely innocuous. I am positive that after the disease has passed away the conjunctiva remains immune for a considerable time, a fact that was at first denied.

If the tiny bacilli are not to be overlooked, the smear must be made at the acme of the disease, when they will be found, as a rule, abundantly between and in the leucocytes.

Treatment of Acute Catarrhal Conjunctivitis

Many aimless things are done to-day in the treatment of acute catarrhal conjunctivitis, but when a physician knows the natural course of a disease he also knows how to treat it. I disapprove of the compresses so often recommended, for it is necessary only to observe once the effect of these dressings to realize that they cannot benefit a disease of the conjunctiva. At the best they merely afford a sort of coolness to the skin of the lid, and that is all. We shall see presently when this cooling may be utilized.

It is imperative that we should first ascertain the etiology of the inflammation by a bacteriological examination of the secretion. If the findings are negative and the condition is due to mechanical or chemical injuries, we must try to deal with these conditions, but in most instances acute inflammations are due to infections.

If the diagnosis of an acute pneumococcal conjunctivitis is established, we know that the inflammation is likely to pass away spontaneously in a short time; hence, we have little to do aside from keeping the eye clean. In graver cases, such as are frequently met with in little children, we inject 10 c.cm. of pneumococcus serum subcutaneously, powder the conjunctiva with the dry serum, and irrigate it with dilutions of the same serum. Friendliness toward this therapy requires that one should frequently see how rapidly the swelling of the lids recedes, even by the next morning, how the children open their eyes and how the secretion is lessened. But the general practitioner cannot practise this treatment, because he hardly has the time or opportunity to make such an etiological diagnosis from the examination of the secretion. Moreover, this specific therapy of pneumococcal conjunctivitis is required only in those cases in which infiltrates or ulcers of the cornea are present. For the second type of this inflammation, that produced by *Koch-Weeks*' bacilli, or by influenza bacilli, we have as yet no specific remedy. We scarcely need them, for acute catarrhal conjunctivitis recovers under other treatment. It is this latter which is needed by both the general practitioner and the ophthalmologist.

When we see that the mucous membrane removes the colonizing microorganisms with its secretion in the spontaneous recovery of such an inflammation, it is self-evident that we should apply no compresses, but should aid the mucous membrane to disburden itself. This we may do by washing away the secretion from the conjunctival sac several times a day with such collyria as, 1, A 3% solution of boric acid; 2, Hydrarg. oxycyanatum, 1:2,000 or 3,000; 3, Kal. hypermanganic., 1:5,000 or 10,000; 4, Hydrarg. chlor. corr., 1:10,000.

Poor patients may prepare the boric acid solution themselves by dissolving from 10 to 15 gm. of boric acid crystals in ½ litre of hot water. With these solutions the conjunctival sac is washed out by the aid of an undine, or eye dropper. Or a piece of absorbent cotton may be wet with the solution and squeezed out gently over the membrane so as to cleanse it. The essential part of this irrigation is the *mechanical* removal of the infectious secretion from the eye. It is not to be imagined that the infected mucous membrane of the eye can be sterilized by such a procedure, or that the inflammation will now cease. Such infections are not overcome by collyria, but by the organism. This mechanical cleansing unburdens the mucous membrane and the patient immediately feels easier.

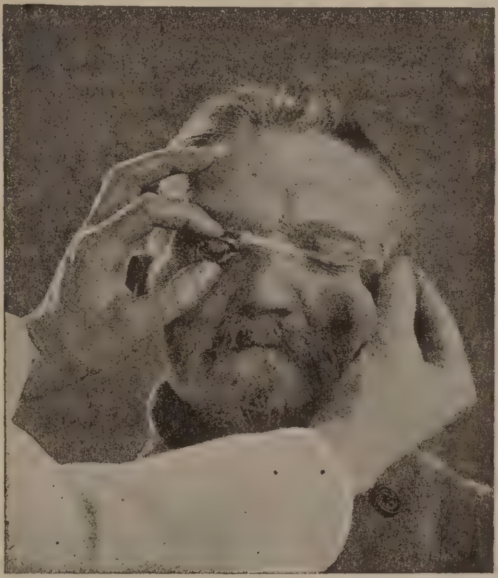

FIG. 11.—Application of Silver Solution to the Conjunctiva.

Our next duty is to destroy the secretion that has been washed out, for every possible endeavor should be made to preserve other persons from infection. Prophylaxis continually accompanies the therapy of an acute infectious conjunctivitis. The margins of the lids and the skin of the face are also to be cleansed with the solution, in order to remove any adherent germs. Included in the prophylaxis of all infectious diseases of the conjunctiva is the admonition that the patient use only his own washbowl, handkerchiefs, etc., and that he keep his hands clean.

These irrigations of the conjunctival sac are repeated several times daily according to the amount of the discharge. When the quantity is large the skin of the lids, particularly about the canthi, may very easily become excoriated; it is therefore wise to dry the skin of the lids after the irrigations and to apply a little vaseline. In the evening borated vaseline should be applied to the margins of the lids that they may not become so glued together during the night that they cannot be opened easily in the morning. Many of the milder cases of acute conjunctival catarrh recover of themselves under such cleansing of the eyes alone, but ordinarily it is necessary to treat the conjunctiva itself. We wish not only to remove the secretion, but to cause it to diminish and finally to cease as the inflammation retrogresses.

The sovereign remedy for this purpose is silver nitrate, the great importance of which was established by *A. v. Graefe*. In acute forms of conjunctival catarrh we apply it in solutions of 0.5%, 1%, and 2% strength, but never in the form of the silver stick, or pencil.

As it is neither suitable nor hygienic to apply one and the same brush to several diseased eyes in succession, we manufacture for each patient his own individual brush. It is quite easy to twist a little cotton on one of the glass rods lying in a solution of sublimate, and the brush is ready. It is imperative that the cornea should be protected while this application is made to the conjunctiva, by holding the everted lids properly. We first evert the upper lid and press its ciliary margin firmly against the eyeball with the middle finger; then at the same time shove down the lower lid with the index finger of the same hand, so as to cause it to evert and thus press together the transition folds so that the cornea is perfectly covered.

A bluish white layer appears on the conjunctiva the moment this wet brush is passed over it. This layer is a crust formed by coagulation of the uppermost layer of conjunctival epithelium by the silver nitrate, a caustic, the effect of which must not be too great. The conjunctiva is rinsed off immediately with physiological saline solution to precipitate at once the free silver in excess. In addition to this we obtain the correct dosage by not taking too much of the silver on the cotton and by not applying it too long; we have no aid here except care and practice.

As soon as the silver has been applied, the eye immediately becomes more irritated, lachrymates more, is more photophobic, and the patient complains of greater pain. This sensitiveness varies a great deal and may be mitigated somewhat, when necessary, by a subsequent instillation of cocain. Such cocainization, however, is to be deprecated, for we wish to produce this irritant effect on the eye. The conjunctival hyperæmia already present is augmented and the mucous membrane becomes more succulent, because an increased transudation has taken place; these signs of greater saturation lead after a time, dependent on the strength of the caustic, to the casting off of the crust. This crust comes away in portions, and as long as any remain the patient will complain of a feeling as if a foreign body were in the eye. The hyperæmia gradually subsides until a feeling of relief appears in from ten to fifteen hours. The most superficial layers of the mucous membrane gradually regenerate, and the first effect of the silver is past in about twenty-four hours.

During this time the secretion of the eye is increased, so it is best to make the application in the morning, otherwise there is apt to be an accumulation of the secretion in the conjunctival sac during the night. The essential point in this treatment is that this increase of secretion is followed by a decrease, which is important as indicative that the disease is passing away. The patient is given a basin of boric acid solution with which to cool his eyes, the one time in the treatment of acute conjunctival catarrh when use can be made of compresses. As soon as the burning pain is past and the sensation as of a foreign body develops, the conjunctival sac is again irrigated.

Several factors take part in the therapeutic action of silver. A large quantity of microorganisms are certainly removed from the eye by the casting off of the sloughing layer of mucous membrane which is full of bacteria. The researches of *Behring*, *Grawitz*, *Martens* and others have demonstrated that silver has a high bactericide power, its action approaching very nearly that of corrosive sublimate, even in the presence of albumin and sodium chloride. But these facts are in no way sufficient to explain the therapeutic power of this remarkable remedy, for the microorganisms can find hiding-places enough in the mucous membrane in which they can escape the bactericide action. Thus, in a blennorrhœa, the sensitive germs resist the remedy for weeks at a time. The principal effect of silver nitrate is indirect. By its use the mucous membrane is affected by a powerful irritant, and responds with a strong reaction; the hyperæmia increases, the diseased tissue becomes still more saturated, and the blood serum can exert more effectively its bactericide and antitoxic powers. The mucous membrane is stimulated and brings about recovery by virtue of its own energies, not directly by the remedy.

As regards the repetition of the application, no hard and fast rules are possible; we must be guided in each case by its gravity and the sensitiveness of the individual. In general, the application should be renewed when the slough comes away, usually after twenty-four hours. If the inflammatory symptoms abate during the course of treatment, the applications may be made at longer intervals, or weaker solutions may be employed.

A word as to contraindications, in regard to which there is much antiquated hypothesis, as *v. Graefe* has said. If silver is not employed when the conjunctiva is secreting a fibrinous, easily coagulating fluid, the nature of the exudation is completely ignored and the remedy is not used just when it will do the most good. Corneal affections not due to the existent conjunctivitis form a rather arbitrary and incorrect contraindication. If the cornea becomes implicated in an acute or chronic catarrh and develops infiltrates or ulcers at the margin, applications of silver to the conjunctiva are directly indicated, for experience has taught us that the reaction thus produced in the eye exerts a favorable influence upon these troubles, even when the corneal ulcer is caused by infectious agents other than those that engender the conjunctivitis. On the other hand we must be careful how we use silver when corneal ulcers develop in the course of a blennorrhœa, even if due to secondary infection. The presence of corneal ulcers in acute conjunctival catarrh is no contraindication to the use of silver. The careful physician will apply the remedy cautiously and in such a manner that the cornea is protected.

Only one other important point remains to be noted concerning silver therapy. When silver nitrate is used for months, or even years, in chronic affections of the eye, the entire conjunctiva, particularly the transition folds, acquire a dirty gray, or blackish brown discoloration. This condition is known as argyrosis. It is due to the fact that combinations of silver gradually diffuse through the tissues and then by reduction deposit the silver in the form of fine granules. Argyrosis is frequently seen in those countries where trachoma is treated year after year with silver. I wish to strongly emphasize this point: No patient should ever take home a solution of silver nitrate. This constitutes the treatment of acute catarrhal conjunctivitis, on the one hand cleansing and the unburdening of the conjunctival sac, on the other the application of silver nitrate.

The attempt has frequently been made to find a substitute for the silver nitrate, which is a harsh remedy in the eye. In the composition of such substitutes emphasis is laid upon the fact that they do not form precipitates with the saline tears, or with albuminous fluids. The hope was that the remedy would sink deeper into the tissues because of this property. Examples of such substitutes are: protargol, a compound of silver and a proteid; itrol, silver citrate; argentamin, a solution of silver nitrate in ethylene diamine; actol, silver lactate; argonin, ichthargan, and others. I have employed these remedies a great deal and my judgment regarding them is as follows: It is true and generally acknowledged that these remedies, especially protargol and argentamin, possess much bactericidal power and are less irritant than silver nitrate. Therefore they are often used. But these qualities alone do not suffice. No matter how strong its bactericide power, no antiseptic can sterilize the infected conjunctival sac. We always need the cooperation of the human economy, we cannot dispense with its stimulation. The fact that all these remedies cause less annoyance is no proof of their therapeutic qualifications in the organism. It is my experience that this therapeutic qualification of the modern preparations of silver is much inferior to that of silver nitrate. In this respect we, like other ophthalmologists, remain conservative, and value the old silver nitrate far above any of the modern compounds.

9. Chronic Catarrhal Inflammations of the Conjunctiva and Their Treatment

In ophthalmology there are diseases the diagnosis of which can be derived in whole, or in part, from the history, and in the recognition of which the description of the troubles plays an important part.

For example, this patient states that for a long time he has been troubled with a sensation of burning and pressure in the eyes; that this is ameliorated when out of doors in the fresh air, but aggravated in a dusty or smoky atmosphere. In the evening in a warm room by artificial light there is a gently beginning sensation of weariness, of heaviness in the eyes. In the morning the eyes are hard to open, there is some secretion in the canthi, and sometimes the lids are glued together.

I. Demonstration of the Various Forms of Chronic Conjunctival Catarrh

These subjective troubles suggest that we have to deal with a chronic conjunctival catarrh. The objective changes in the mucous membrane have now to be noted.

1. The Graver Form of Chronic Conjunctival Catarrh

The chronic forms of conjunctival catarrh are differentiated from the acute, first by the fact that the secretions are much less in quantity. In the present case only a little frothlike matter is visible in the canthi. The secretion is usually thicker and more glutinous, and dries firmly on the margins of the lids. The mucous membrane is no longer as smooth and glistening as it is under normal conditions. It is not so infiltrated with serum as in acute catarrh, but its surface is uneven, a condition particularly obvious in the vicinity of the transition folds, where the conjunctiva has a velvety appearance, due to a greater development of the papillary bodies.

The lids of patients with chronic conjunctival catarrh frequently have red margins. The intermarginal space, as well as the neighboring portion of the skin, is reddened and somewhat thickened. Excoriations, cracks and little fissures are often found near the canthi. Changes of this sort are the easily comprehensible sequelæ of the action of the conjunctival secretion in prolonged contact with the skin, which leads to a softening of the epidermis.

When the chronic conjunctivitis has existed for a long time it occasions a change in the position of the lower lid. In this case the inner portion of the lower lid, containing the lower punctum lacrymalium, is dragged away somewhat from the eyeball and turned slightly outward. This is spoken of as a commencing ectropion of the lower lid. It is worth noting that this turning outward of the lower lid in a chronic conjunctival catarrh begins by preference at the inner canthus. When this takes place the condition frequently grows worse, the aggravation being due to the following factors:

The drainage away of the lachrymal fluid is interfered with by the eversion of the lachrymal punctum, and the fluid collects in the conjunctival sac, where it becomes mixed with the conjunctival secretions and furnishes a suitable medium for the inflammatory agents present, which increases the catarrh.

When the tears are not properly drained away, the skin of the lid is kept constantly wet by the epiphora and a dermatitis angularis readily develops.

The inflamed conjunctiva keeps on becoming thickened by papillary hypertrophy, for the eversion of the lid exposes it to the influences of the air, wind, smoke and dust, which are very irritating. The swollen conjunctiva tarsi then presses the palpebral margin still farther from the eyeball and increases the ectropion.

Still other factors may be added. In many cases the skin of the lower lid becomes continually harder and more inelastic because of the dermatitis, and the thickened skin increases the ectropion. In other cases, especially in old people, the orbicularis and the skin of the lid become relaxed, and this leads to a sinking of the lower lid. Then when the patient wipes the tears away, he draws the lower lid from the eyeball if he wipes downward. Thus chronic conjunctivitis starts a vicious circle which necessarily produces these results. In other grave cases the wet eczematous skin of the lid is shortened horizontally and produces a narrowing of the palpebral fissure, blepharophimosis.

2. *Less Serious Cases of Chronic Conjunctival Catarrh*

Mild forms of chronic conjunctival catarrh are very common. Many persons have such a condition unknown to themselves. In these patients the slight redness is confined to the conjunctiva tarsi, there is almost no secretion; at the most we find only a little at the inner canthus in the morning. This form is known as conjunctivitis sicca, or catarrhus siccus. The cardinal symptoms of a catarrhal conjunctivitis, redness, swelling and abnormal secretion of the conjunctiva, are present only in a very slight degree. The swelling particularly is so slight that the *Meibomian* glands may be distinctly observed beneath the conjunctiva.

3. *Case with Slight Follicle Formation*

In addition to the changes that have been mentioned, isolated follicles may develop in the lower transition fold in some cases of simple chronic conjunctival catarrh. They are usually small, and remain sharply differentiated from the surrounding tissue without much redness. Such follicles may be found also in the outer and inner corners of the upper transition fold, though no trachoma is present. This fact should be borne in mind, as it will be touched upon in considering the differential diagnosis of trachoma.

II. Etiology of Chronic Catarrh of the Conjunctiva

One of the commonest forms of chronic conjunctival catarrh is the diplobacillus conjunctivitis, which will be considered more in detail later, as it demands a certain therapy, and is so common as to justify its separate consideration.

When dealing with a chronic conjunctivitis it is wise to ascertain first whether it is a case of diplobacillus conjunctivitis. This is done by a bacteriological examination.

The bacteriological findings in chronic conjunctival catarrh are not apt to be of much importance, except in the case of diplobacillus conjunctivitis, and when this is excluded the question arises as to its origin. Mankind is born of and lives in dust. No proof is needed to show that dust, smoke and vitiated air affect the eyes; any one who has a chronic conjunctival catarrh knows that his troubles are aggravated by such influences. Numerous artisans suffer from a chronic catarrh which is in part an occupational disease. Continuous exposure to wind and weather causes chronic conjunctival catarrh in farmers and coachmen. The mucous membrane of the human eye bears exposure to the atmosphere badly, so a chronic catarrh of the conjunctiva may lead to ectropion, and inversely, an ectropion from some other cause produces irritation and chronic inflammation of the conjunctiva. We also find signs of chronic conjunctivitis in laborers whose eyes are exposed to high degrees of heat.

Attention has recently been called to the fact that the short waved rays of light may irritate the conjunctiva. Many ophthalmologists ascribe so chronic a disease as vernal catarrh to the action of the ultraviolet rays. In all such cases the eyes are affected by chemico-physical stimuli.

Chronic conjunctival catarrh may also be due to many other causes, some of which are to be found in the eye itself. An extremely frequent cause is an obstruction of the lachrymal duct. Particularly when one eye only is affected pressure upon the lachrymal sac should be instinctively our first move.

When an adequate cause cannot be discovered in the lachrymal passage we should examine the margins of the lids, from which the conjunctiva is very easily affected. A pustulous eczema and a sycosis of the margin of the lid is always accompanied by a conjunctivitis; desquamating eczema, and seborrhea sicca are almost always associated with a very obstinate form, and *Peters* has described a peculiar form of chronic conjunctivitis met with in people the skin of whose lids is abnormally soft and exhibits minute fissures.

Finally, the causes may be situated in the conjunctiva itself. We find very frequently, even in young persons, small, whitish-yellow, hard concrements in the conjunctiva tarsi, calcareous infarcts of the *Meibomian* glands, which maintain a chronic state of irritation of the conjunctiva in their immediate vicinity. It is also necessary to notice whether a chalazion is present, either in its initial or a later stage, for these likewise affect the conjunctiva.

In addition to all these local causes it must be remembered that irritants in the blood-stream, due to general diseases, may excite a conjunctivitis. It is true that these inflammations are usually acute in their nature and derived from such infections as measles, scarlet fever, influenza, or gonorrhœa, but there is a disorder of metabolism which is often accompanied by a chronic conjunctivitis having periodic exacerbations, the uric acid diathesis. It is well to bear this possibility in mind.

When all of these causes have been excluded there still remain the anomalies of refraction, especially hypermetropia and astigmatism, which are competent to maintain a condition of congestion in the mucous membrane of the eye. This is a factor which must never be neglected. The asthenopia, which is associated with uncorrected anomalies of the refraction, leads to an increase of the innervation of the eyes, this to a functional hyperæmia of the conjunctiva, which, finally, if long continued, causes permanent changes in the margin of the lid and the mucous membrane. This connection is not one to be underestimated. If a patient goes to an ophthalmologist in America, or in England, he receives almost invariably a prescription for glasses, even if there be only .25 D of astigmatism to correct. On the other hand conditions are frequently such that a chronic conjunctivitis may excite an asthenopia. Therefore, it is wise to proceed cautiously in the matter of therapeutics. Nevertheless anomalies of the refraction certainly play a part in the chronic hyperæmia of the conjunctiva.

The course of a chronic conjunctival catarrh is very tedious. Many persons suffer more or less all their lives from this trouble. Aged people have almost invariably a catarrhus senilis. In all forms the trouble is at times better, at other times worse.

Anatomically, we find an increase of the adenoid tissue, the prominent papillary elevations. The increase of the goblet cells in the epithelium is particularly striking.

III. Treatment of Chronic Catarrhal Conjunctivitis

When a physician has arrived at a diagnosis of chronic conjunctival catarrh he ordinarily prescribes a ¼ to ½% solution of zinc sulph. to be instilled. In many cases this is indicated, and it is particularly beneficial in a chronic conjunctivitis caused by diplobacilli, but the variable etiology indicates that in many cases further investigation is necessary and that the cure is not easy, but in some cases impossible.

In making the diagnosis of a chronic conjunctival catarrh, we naturally start with consideration of the eye and end with the functional test, but in treatment this order is reversed; we begin with the correction of whatever functional disturbance may be present. If we find an error of refraction with hypermetropia or astigmatism, we first prescribe suitable glasses; for we know that they are apt to be accompanied by congestion of the conjunctiva. Then we take into account the environment of the eye, looking first for any changes in the skin and margin of the lid, which must be cared for from the commencement of treatment. If trouble in the lachrymal passage is the cause, it must be obviated.

An important clinical fact, the truth of which has often been demonstrated, is that chronic conjunctival catarrh improves rapidly after the removal of chronic intranasal troubles. For this reason obstinate cases should be subjected to a rhinologic examination.

In the treatment of the conjunctiva itself, it must be borne in mind that people with chronic conjunctival catarrh often must be first taught how to take care of their eyes. We prescribe at first for cleansing of the eyes and margins of the lids one of the following solutions: boric acid solution, 2 to 3%; borax solution, 3%; hydrarg. oxycyanat., 1 : 5,000; sublimate, 1 : 10,000. We do not expect to sterilize the mucous membrane with such collyria, for they are too weak, and stronger solutions would injure the tissues. Clinical observation has shown that astringents, rather than antiseptics, are to be preferred in the treatment of chronic conjunctivitis, as the mucous membrane of the eye responds better to astringents with a reaction through which protective bactericide substances are caused to enter the secretion; so next we have to choose what astringents to employ.

Contrary to our custom in acute catarrhal inflammation, we do not use silver. Weak solutions are very easily disintegrated, and the nitric acid thus set free is then very irritating, while the silver ions, which unite with the albumin to form an albuminate, are toxic to the cells. It is not uncommon to see a chronic conjunctivitis improve as soon as the application of silver is discontinued. When it is desirable to employ a preparation of silver, a 1 to 5% solution of protargol is preferable.

Zinc is used in the form of the sulphate, or sozoiodolate, in ¼ to ½% solution.

Another very useful remedy in simple chronic conjunctival catarrh is the collyrium astringens of the Austrian Pharmacopeia. We direct the eyes to be washed out with the prescribed lotion immediately on arising in the morning, instill a drop of the astringent collyrium after breakfast and again toward evening. Just before retiring for the night the eyes are again washed out, or an ointment is applied. In many cases, especially those in which single follicles are to be seen, the alum pencil is of good service.

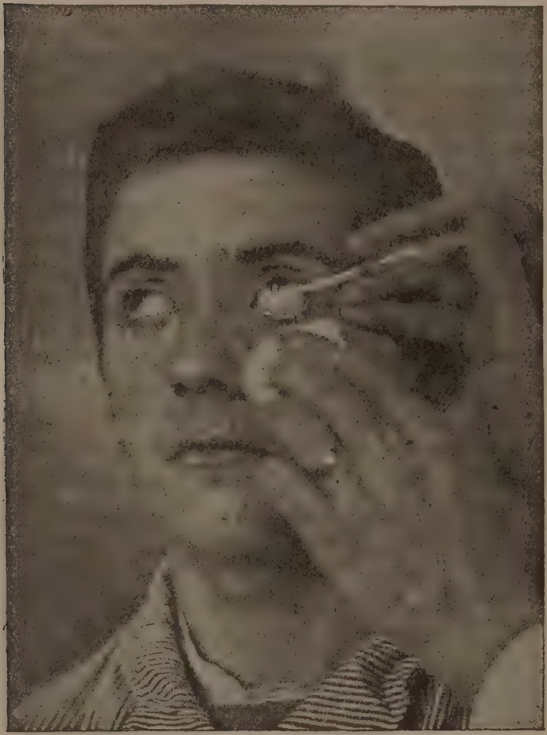

FIG. 12.—Instillation Into the Conjunctival Sac.

The effect of adrenalin, a remedy much used of late, is striking. We need only a few drops of a 1:1,000 solution to render almost immediately pallid the hitherto red mucous membrane, through its marked power to contract the vessels. Unfortunately, this effect is very transient; but, nevertheless, the drops are very popular among the better class of patients, and we may add some drops of a 1:1,000 solution to the collyrium.

Cocain, on the contrary, causes a loss of sensitiveness in the eye, and its continued use in chronic conjunctival catarrh is to be deprecated. It also shortens the life of the epithelium.

As the *Meibomian* glands in very many cases contain abnormal quantities of secretion, it is highly advisable to vigorously massage the margins of the lids and to express the secretion. After cocainization a thick, oiled, glass rod is passed beneath the lid, and the margin of the lid is pressed firmly upon it with the thumb, causing the whitish secretion to exude in great masses. The glands are disburdened as we pass systematically along the entire margin. In this way the redness of the margins of the lids may be caused to quickly disappear. Still obstinate troubles sometimes remain after the papillary hypertrophy has disappeared. These are particularly marked in catarrhus siccus. In the most obstinate cases it occasionally happens that the upper lid lies too tensely on the eyeball, and we may be obliged to enlarge the palpebral fissure; but this is done in simple catarrh only in case of extreme necessity. In other cases, the mucous membrane has become too dry. In these troubles daily massage of the conjunctival sac with the zinc-ichthyol ointment recommended by *Peters* may give excellent results. The prescription is:

Ammon. ichthyol	0.15
Zinc oxid	5.0
Vaselin alb	15.0

M. f. exactissime ferendo ungt.

If the trouble does not yield to this, we may obtain help from two little operative measures. After cocainization, we may evert the lid and remove the epithelium from the tarsal surface with a scarificator. This procedure is followed by a slight irritation and increase of the secretion, and very often after a few days a quite remarkable amelioration is produced.

When there is a greater degree of tumefaction of the conjunctiva, we may make with a small knife numerous little incisions in the hypertrophic mucous membrane parallel to the margin of the lid, so as to cause a profuse bleeding. Not infrequently a substantial improvement is obtained in this way which may last for months.

Finally, we find, together with the chronic conjunctival catarrh, a marked sensitiveness of the supraorbital nerve, from which severe pains radiate, particularly in sensitive people. In such cases *Peters* recommends the internal use of quinin and iron in combination with secale cornutum:

Secal. cornut.	1.0
Quinin. muriat	2.0
Ferri sulph	3.0

Extr. gent., q. s. ut f. pil. No. 100.
Sig.: Two pills three times a day;

or ½ to 1 teaspoonful of tinct. eucalypti globuli may be taken in a glass of water after meals.

Asthenopic troubles may also be helped in this way when no other cause is apparent than an oversensitive condition. It is of considerable importance that the digestion be regulated, and it scarcely needs to be mentioned that anæmic conditions and scrofulosis must be cared for. Many patients with chronic conjunctival catarrh are relieved in this way. But there will still remain many cases, the etiology of which cannot be elucidated and where treatment fails to effect a permanent cure. The advice to avoid smoke and dust is easily given. In many instances we are obliged to content ourselves with some amelioration of the troubles which constantly recur.

10. The Diplobacillus Conjunctivitis: One of the Chief Forms of Chronic Catarrhal Conjunctival Inflammation

The history of our patient indicates a slight catarrhal trouble. The important point is that this trouble has lasted a long time, for many months. This is a form of chronic conjunctivitis which, if not treated, may persist for several years. It is caused by infection with the *Morax-Axenfeld* bacillus, and is called diplobacillus conjunctivitis.

The Clinical Syndrome of Diplobacillus Conjunctivitis

The margins of the lids are red, but only at the canthi. The color is most marked at the inner canthus, where it is of a peculiar livid hue. The skin in the reddened canthi is moist, appears to be somewhat macerated, and is covered by a gray-yellow, tenacious secretion. The changes in the conjunctiva are insignificant as compared with those at the canthi; it is somewhat reddened as far as the transition fold, but the conjunctiva bulbi is hardly involved, except that it is rather red in the vicinity of the canthi. This is a chronic catarrhal inflammation of the conjunctiva, located chiefly at the canthi. It has been called quite properly blepharoconjunctivitis angularis.

The conjunctival secretion is very slight, but if we take a smear of the mucus lying on the caruncle, we will find almost invariably numerous short, plump bacilli, usually in pairs, in the secretion which ordinarily contains few cells. When epithelial cells are present they are usually densely covered with bacilli. Phagocytosis is rare. The microorganisms have rounded ends, vary in size and are often in the form of small diplobacilli. They are easily recognized; they are decolorized by *Gram's* method and have only a slight capsule formation.

The culture of this pathogenic agent is quite as characteristic as the slides. It grows on blood serum, serum agar and ascites agar only at the temperature of the incubator. The cultures on blood serum are very typical. Where the colonies develop deep cavities are formed, for the diplobacillus liquefies the solid blood serum. This and the related *Petit's* diplobacillus are the only morbid agents among the bacteria of the conjunctiva that possess this characteristic. *Petit's* diplobacillus may be disposed of in a few words. It can be differentiated from the common *Morax-Axenfeld* diplobacillus only by the fact that it grows more luxuriantly and on ordinary agar. It belongs to the same group of morbific agents, for it has been proved able to produce the picture of diplobacillus conjunctivitis in man. It is met with rarely in conjunctival troubles, but is comparatively frequent in the cornea.

These diplobacilli are able to retain their vitality for a long time outside of the body in a dry condition, particularly in dried secretion. They have also been found in the nasal tract of healthy persons by *Erdmann*, so we must suppose that the eyes may become infected through the nasal secretion. According to the researches of *Morax*, *Axenfeld* and others, the inflammation begins after an incubation period of four days in the conjunctival sac. The diplobacillus is not pathogenic for animals, but man is extremely susceptible to this infection and the inoculation of the secretion almost invariably results in the disease. The diplobacillus has been met with on the healthy conjunctiva only in rare instances. Once settled on the human conjunctiva, it continues there very long, if the inflammation is not treated. Attention is called to this disease for two reasons.

In the first place this disease is very often not recognized, because its objective symptoms frequently are very slight. When, however, we have learned to look for the characteristic changes in the canthi, we find that this diplobacillus conjunctivitis is a very widespread disease. I can only advise that when a patient comes to be treated for an obstinate chronic conjunctival catarrh this diplobacillus conjunctivitis should be considered primarily.

In the second place it happens very often that the physician torments himself in vain with the treatment of a conjunctival catarrh, and all our usual remedies are impotent in a diplobacillus conjunctivitis. The consensus of opinion commends preeminently in such cases treatment with zinc.

Treatment of the Diplobacillus Conjunctivitis

We first wash out thoroughly the conjunctival sac with a weak solution of zinc. Then we prescribe zinc sulphat., 0.03, aquæ destill. ad. 10.0, to be instilled regularly at certain intervals. Finally, the patient is instructed to apply a zinc ointment of the same strength to the eye in the evening and, particularly, to smear it on the inflamed canthi. Why the effect of zinc on the diplobacillus conjunctivitis is so good, has not yet been learned. It is not directly bactericidal, for the experiments of *Paul*, *Silva* and others have proved that the diplobacillus is very resistant to solutions of zinc.

If our patient is to be freed from his troubles he will need to continue with this zinc therapy for weeks at a time, for otherwise relapses are frequent, and I cannot agree with those who speak of zinc as a "specific" for diplobacillus conjunctivitis. Very obstinate cases have been met with in which consistent treatment with zinc has proved unavailing. In such cases I have finally succeeded by the aid of active immunization.

Ordinarily, no immunity to the disease is acquired; we see the trouble last for years, we see relapses occur; it may be after an original cure with zinc, it may be after experimental reinfection. I have therefore made cultures of the diplobacilli met with in such persons and, after killing the cultures, have injected them in increasing quantities subcutaneously. The result was then quite satisfactory, the zinc therapy being continued. The obstinate inflammation definitely vanished, and artificial infection with a fresh culture produced no effect. But this is a form of treatment which can be carried out only in the clinic. Elsewhere treatment with zinc must suffice, and in most cases it is competent.

11. Conjunctivitis Pseudomembranosa and the Value of Diphtheritic Serum in Diphtheritic Infection of the Eye

This child has a severe inflammation of both eyes. His physician states that on the first day it appeared to be an acute catarrhal affection, but that it assumed such a threatening character on the second day that he advised the child to be sent to the clinic. The lids of both eyes are red, swollen and hot. The tumefaction of the upper lids is particularly marked; they are greatly infiltrated and feel almost as hard as boards; the child cannot open his eyes.

Although at first glance this condition recalls the grave symptoms of a gonorrhœal infection still, in contrast to the phenomena presented in that disease, there is no blennorrhœa, no pouring forth of pus from between the lids. At present the secretion is very moderate in quantity. We will now separate the lids with a retractor.

The entire surface of the conjunctiva of the lower lids, even up to the transition fold, is covered by a yellowish gray, or dirty gray, opaque membrane. In the lower transition fold, which alone can be brought into view, the mucous membrane is yellowish white, fatty and studded with red points scattered over it. In graver cases this necrosis may sink deeply into the tarsus, and even the neighboring skin of the lid may become partially, or wholly gangrenous. Other serious cases may show isolated necrotic islands in the skin of the lid. The conjunctiva bulbi is livid, yellow, chemotic and surrounds the cornea like a rigid wall. Now, while the lids are held apart, a small quantity of thin, turbid fluid wells up from the palpebral fissure; it is stained red because the infiltrated and membrane-covered conjunctiva bleeds on slight provocation. If a diagnosis is desired now from the clinico-anatomical view point one can say only that this is a conjunctivitis associated with a formation of membrane. But this is not of the least help to us, or to our patient.

It is true that formerly a clinical distinction was made between a superficial, mild form of this disease, which was called conjunctivitis crouposa, and a graver form known as conjunctivitis diphtheritica, and it would be difficult to find another form of conjunctivitis in which the advance of science could be so clearly demonstrated as in this conjunctivitis crouposa, for modern bacteriological research has proven that the clinical picture of a conjunctivitis associated with the formation of a membrane may be produced by any one of a long list of very different pathogenic agents.

Here we encounter the biological law, previously mentioned, which declares that the manner in which the mucous membrane of the eye reacts to an infection depends on the predisposition, or susceptibility, of the body as a whole, on the local condition of the conjunctiva itself, and on the peculiarities and virulence of the morbific bacterial agent concerned. It has been demonstrated that croupous inflammation of the conjunctiva may be excited by gonococci, pneumococci, streptococci, diphtheria bacilli, *Koch-Weeks*' bacilli and other micro-organisms. The mildest case of conjunctivitis crouposa may be caused by very virulent diphtheria bacilli, the gravest diphtheritic conjunctivitis by streptococci, and vice versa. Therefore the clinical differentiation between croupous and diphtheritic conjunctivitis is unsatisfactory. It is better to designate this entire group of inflammations of the conjunctiva as conjunctivitis pseudomembranosa. It is essential to bear in mind the fact that the formation of a pseudomembrane may be a symptom evolved from an infection by bacteria of the most various species.

Even the differentiation formerly emphasized, that after recovery from conjunctivitis crouposa the conjunctiva regained a perfectly normal condition, while conjunctivitis diphtheritica resulted in necrosis of the infiltrated parts and in the formation of cicatricial tissue, can be maintained no longer. A severe streptococcal infection of the conjunctiva, presenting the clinical aspect of conjunctivitis crouposa, may result in a considerable gangrene of the conjunctiva and large cicatrices, while a mild infection with diphtheria bacilli may run its course without injury to the conjunctiva.

It is also incorrect, pathologically, to state that in conjunctivitis crouposa the membranous formation is merely superficial, while in conjunctivitis diphtheritica the exudation is deeply rooted in the tissue. We meet with many intermediate forms, and it has been demonstrated that both forms of exudate may be met with on one and the same conjunctiva. It is, I think, a step backward when some of the elder ophthalmologists continue to adhere to a single concept, to a superannuated clinical system, and to persist in the differentiation of conjunctivitis crouposa from other forms of conjunctival inflammation, particularly from that known as conjunctivitis diphtheritica. The modern and correct view governing us in every case of conjunctivitis associated with the formation of membrane, and in accordance with which we have proceeded in our work, is as follows:

Two things are a priori possible in a case of conjunctivitis associated with the formation of a membrane:

1. The formation of a membrane may be due to a traumatism, which may be of a mechanical, chemical, or thermic nature.

Thus, after operations on the conjunctiva, e.g., after operations for strabismus, a wound may become covered by a fibrous coat. A foreign body may be a source of irritation in the conjunctiva for a long time and give rise to a fibrinous excretion. The action of acids, alkalies and other chemicals may be followed by the formation of a membrane. Typical examples are the membranes met with after lime burns of the conjunctiva and those that follow the therapeutic application of silver. Jequiritol may induce a typical conjunctivitis crouposa, as has been stated already. Likewise it has been shown that burns of the conjunctiva with red hot metal may be followed by the formation of a membrane. When the etiology is of this nature it is learned immediately from the history, and we need to devote no more space to it.

2. The formation of membrane may be consequent upon an infection. Then we inquire into the nature of the disease, for the clearing up of the etiology furnishes the necessary indications for proper treatment. First I shall state the measures that we can use in the clinic, and then sketch what the practitioner should do in such cases. Smears are prepared from the membrane, in which may be found not a few *Gram* positive bacilli, clubbed at the ends, or dumbbell shaped. As soon as these are seen the child should receive his first injection of diphtheritic serum. The smears should be made as thin as possible, and several must be examined when the finding is at first negative.

Still we are unable to decide positively from the smear alone that the infection is one of diphtheria. For you know from the study of the bacterial flora of the conjunctival sac that a bacterium, called the xerosis bacillus, is normally present in every conjunctiva, and that it is very similar morphologically to the diphtheria bacillus. Cultures must therefore be prepared by smearing the material on agar, or serum agar, and transferring from this an inoculation into *Lœffler's* serum.

A word only concerning the differential diagnosis between the true diphtheria bacillus and bacteria resembling it, chiefly the so-called pseudodiphtheria bacillus and the xerosis bacillus. The pseudodiphtheria bacillus forms little or no acid and is avirulent for guinea-pigs, and the different varieties may also be distinguished by agglutination, but neither of these methods furnishes a positive diagnosis, as there are numerous transition stocks.

The question has also been raised whether the xerosis bacillus is not an avirulent diphtheria bacillus. I agree with *Axenfeld* that there is no evidence that the xerosis bacillus is able, or can be made able, to produce true diphtheria toxin. According to *Bietti* animals inoculated with xerosis bacilli were not immunized against the virus of diphtheria. In the second place in experimental inoculations with xerosis bacilli nothing can be recognized which indicates in any way the production of diphtheria toxin. The theory advanced by *Behring* that the xerosis bacillus may occasionally assume a virulent form and cause the milder diseases known as diphtheroid has not yet been demonstrated to be correct. But if, with *Lœffler*, we consider identical with the diphtheria bacillus only such bacilli as are able to produce the specific toxin we are able to isolate the diphtheria bacillus from among bacteria that are very similar morphologically.

After some hours the cultures have begun to grow and, if we find in them colonies typical of the diphtheria bacillus, we use first *Neisser's* granule stain (acetic methylen blue—Bismarck brown). *Neisser* made the discovery that by means of this stain a characteristic granular condition is produced at the poles of the true diphtheria bacillus in from nine to twenty hours, and when we examine the smear thus stained we can see the characteristically placed, slender, yellowish brown rods with beautiful, oval, black blue granules in their poles. A positive result of this nature informs us within a few hours after the clinical examination of a patient that the infection is very probably, if not certainly, due to diphtheria bacilli. Absolute certainty may be attained through proof that the bacilli found are able to form the specific diphtheria toxin.

This toxin may be demonstrated first by animal experiment, second, by neutralization of it by means of the specific antitoxin. The first suffices to establish the diagnosis. A tube of bouillon is impregnated from numerous characteristic colonies, in order to avoid the chance that bacilli might be taken from a single non-virulent colony, and allowed to stand for twenty-four hours. Two cubic centimetres of this bouillon culture are then injected beneath the skin of the abdomen of a guinea-pig. The animal dies of diphtheria in from two to four days, after the development of a doughy swelling of the subcutaneous tissues at the place of injection, surrounded by a brawny and hemorrhagic area. The suprarenal capsules will be characteristically reddened and a serous exudate will be found in the pleural cavity, often in the pericardial sac as well. But the treatment of our patient must not be delayed until the result of this experiment is known. I do not consider it right that we should await the result of all these investigations, even in cases that appear to be mild in character, because we can never know whether the activity of the toxin is not preparing the way for secondary infections, and therefore it is imperative that we should deal as quickly as possible with the fundamental disease and inject the diphtheria serum as soon as we have good reason to suspect the presence of diphtheria.

The beneficial effect of diphtheria serum in diphtheria of the conjunctiva is acknowledged everywhere, by a rare and happy consensus of opinion among ophthalmologists. That its effect can be certain in a diphtheritic infection alone is a matter of course, when we bear in mind the law of specificity of the immunity reaction between toxin and antitoxin. Its effect is made manifest in a short time by the melting away of the membrane and the disappearance of the new formation. It also prevents the spread of the disease to other mucous membranes. The earlier in the course of the disease the injection is given the more certainly will a good result be produced.

It may be asked how much antitoxin it is necessary to use to produce this curative effect on the eye. The elaborate studies I have made concerning the neutralization of the diphtheria toxin by the antitoxin in the subconjunctival tissue and the cornea have convinced me that the number of units which have usually been employed in diseases of the eye is too small, that a single dose of 1,000 units is not sufficient. Larger doses should be given. In addition to the subcutaneous injection of antitoxin a powdered, dry diphtheria serum may be dusted over the conjunctival surface, so that the antitoxin is brought into direct contact with the toxin and will contribute to its neutralization.

The croupous inflammations of the conjunctiva are almost exclusively diseases of childhood, and we know from clinical experience that all of the infections which cause pseudomembranous conjunctivitis seize by preference upon feeble and ill-nourished children who are convalescent from measles, or some other infectious disease. The only disease from which this condition needs to be differentiated is the very rare acute pemphigus, but in pemphigus the membranes are easily removable and recur for months. Ulcerative diseases of the conjunctiva with a fibrinous coating are easily recognized. The appearance of granulating ulcers is very different, and they do not have the same acute symptoms at first that are present in diphtheria.

In benign cases the swelling of the lids recedes, the folds in the palpebral skin reappear, the tissues soften so that the lid can be everted, the secretion increases at first and then diminishes gradually until the conjunctiva has regained its normal condition. In severe cases only, the conjunctiva is partially destroyed by necrosis and cicatrices are formed. Gangrenous inflammations may result in a shortening of the palpebral fissure, ectropium, symblepharon, trichiasis, and xerosis. So far as such grave forms depend on the pure diphtheria infection they have become very rare since the introduction of serum treatment.

The involvement of the cornea forms one of the most important considerations throughout the entire course of conjunctival diphtheria. The experimental studies of *Coppez*, *Dugast* and others have demonstrated that after prolonged instillation of diphtheria toxin into the conjunctival sac the cornea may become opaque and necrotic. I have found that swelling, opacity and partial necrosis of the tissue may be occasioned by the injection of a few drops of a 1:10,000 dilution of diphtheritic virus into the parenchyma of the cornea. These changes have been studied by my assistant, Dr. *Grueter.* According to *Uhthoff*, the diphtheria bacilli themselves cause a purulent infiltration in the corneæ of animals. In man the conditions are such that the toxin may produce a pseudomembrane, but it rather prepares the way for secondary infections. Suppuration of the cornea induced by diphtheria bacilli is exceptional in conjunctival diphtheria; it is usually the result of secondary infection with pus germs. It may start in the middle, or at the margin of the cornea and is not entirely due to the interference with nutrition by the diphtheritic infiltration of the conjunctiva, as was formerly thought to be the case. Serum treatment exerts no such certain influence over the corneal complications when they are due to secondary infection as it does over the conjunctival trouble.

It must be borne in mind that the infection of conjunctival diphtheria is a source of danger both to the patient himself and to others. The disease may extend from the conjunctiva to the mucous membranes of the nose, throat, larynx and trachea, and, in malignant cases, cause death, but when the serum has been administered in time, conjunctival diphtheria usually follows a benign course. Careful precautions must be taken that the infection may not be communicated to other persons by the patient. The patient must be isolated and sometimes prophylactic immunization of the other members of his family will be necessary.

Conjunctivitis Pseudomembranosa from Other Causes

It has been stated already that the same clinical picture of conjunctivitis pseudomembranosa may be produced by other pathogenic agents than the diphtheria bacillus. Such agents are the gonococcus, the *Koch-Weeks'* bacillus, the staphylococcus, *Friedlænder's* pneumonia bacillus, the bacterium coli, preeminently the pneumococcus and the streptococcus, and we must consider what our conduct should be in such cases, particularly how the general practitioner, from whom such an accurate etiological diagnosis cannot be expected, shall deal with them.

When the infection is produced by a microorganism against which we can interpose no such certain remedy as the diphtheria serum we must content ourselves with the measures employed in other infectious inflammations of the conjunctiva: cleansing, protection of the other eye from infection, and washing out of the conjunctival sac. As soon as the membranes have been cast off they should be removed carefully from the eye. No application of a caustic nature should be made to the conjunctiva while the inflammation is in this stage, but after the formation of membranes has ceased, the conjunctival secretion has become greater in amount and the lids have become movable, the application of a one per cent. solution of silver nitrate is indicated. Cold should never be applied to diphtheria of the eye, because the infiltrated tissue is already suffering from impairment of nutrition and has a tendency to necrosis. When the cornea is involved treatment must be confined to the introduction of an ointment containing either iodoform, or bichloride of mercury in the strength of 1 to 10,000, into the conjunctival sac.

The situation is somewhat different when we have to deal with a croupous conjunctivitis due to a pneumococcus infection. Many such infections run a harmless course, but occasionally the pneumococcus excites a very grave pseudomembranous conjunctivitis, as in cases reported by *Becker*, *Kimpel*, *Hertel*, *Roscher* and others. In such cases it is advisable to administer large doses of pneumococcus serum both subcutaneously and locally, for in my experience it has proved very efficient.

Streptococcal infections deserve special consideration, for the streptococcus is the pathogenic agent which, either alone, or in association with the diphtheria bacillus, is most frequently responsible for the production of a pseudomembranous conjunctivitis. The most virulent forms of this inflammation, in which the conjunctiva becomes necrotic and the cornea destroyed, are the work of this agent. The children attacked are, as a rule, badly nourished, suffering from hereditary syphilis, or convalescent from some acute disease. Great numbers of these cases have been described in medical literature. It is not a matter of surprise that many of these patients die of secondary sepsis, when we consider how the conjunctiva and cornea have been changed into a dense culture of streptococci. In the majority of the cases of croupous conjunctivitis following measles, or scarlet fever, the pathogenic agent is the streptococcus, and yet it is exceptional to meet with the streptococcus in a case of simple conjunctivitis.

Stenosis of the nasolachrymal duct is associated with a conjunctivitis excited by streptococci, that has been named lachrymal streptococcal conjunctivitis. These cases are frequently complicated by an irritation of the iris caused by the resorption of the toxin.

Finally streptococci have a certain predilection for the conjunctivitis that so frequently accompanies an eruption of impetigo on the face in persons of a scrofulous diathesis.

In all serious streptococcal inflammations of the eye recourse should be taken immediately to the specific streptococcus serum. The longer my experience with this serum the stronger my conviction becomes that it cannot be denied an action comparable with those of other serums. The difficulties that militate against the development of its therapeutic effect lie in the facts that streptococcal serums from different stocks differ in efficiency, and that it is commonly employed too late. But many times it has proved itself able to save not only the eye, but the life of a child suffering from a grave streptococcal infection and it must therefore not be left untried.

It is well known that the streptococcus is met with as the agent of infection very frequently in other parts of the body, so it is well to emphasize the fact that it appears in the eye with comparative rarity. When met with in the eye a differential diagnosis from the pneumococcus is always necessary. This may be made by the experimental inoculation of a mouse, because the pneumococcus will then appear encapsulated in its blood.

Finally, what shall the general practitioner do in view of the manifold etiology of pseudomembranous conjunctivitis and these difficult questions in bacteriology and differential diagnosis? In any doubtful case the subcutaneous injection of diphtheria serum can do no harm, for none of the other germs are of equal importance with the diphtheria bacillus, but grave cases should be handed over to the specialist for treatment.

12. Conjunctivitis Gonorrhoica of Infants (Blennorrhoea Neonatorum) and Its Clinical Course

It needs but a glance at an infant a few days old, with streams of thick yellow pus welling forth from between the lids of both eyes, to make the diagnosis of blennorrhœa neonatorum, a grave disease of the eye caused by the gonococcus discovered by *Neisser*. In reference to this disease there are five things with which every physician should be conversant: I, its clinical picture, II, its pathogenic agent, III, its course and prognosis, IV, its treatment, V, its prophylaxis.

I. The Clinical Picture of Blennorrhoea Neonatorum

The lids of both eyes are very red and so swollen that they can scarcely part sufficiently to allow the pus to gush out from time to time through the palpebral fissure. Measures must be taken to examine the eyes more closely, and in attempting to do this it must be borne in mind that this disease is extremely contagious. It is not an infrequent occurrence that a physician infects his own eyes while making this examination and I was once obliged to enucleate the eye of a colleague that had been destroyed by such an infection. The physician should protect his own eyes, by means of large glasses, against the danger of any secretion being spattered into them, just as he does in the treatment of trachoma. He then places the child on his lap in such a way that the head rests between his knees, lying on a towel, while the nurse holds the hands. Larger children may be held in the same manner, the head gripped between the physician's knees, the arms and legs controlled by the mother, or a nurse, and examined satisfactorily even when they are photophobic, or are struggling to prevent the examination. The next important point in the examination of a case of ophthalmia neonatorum is that the pus must not be allowed to spirt when the lids are opened. A piece of

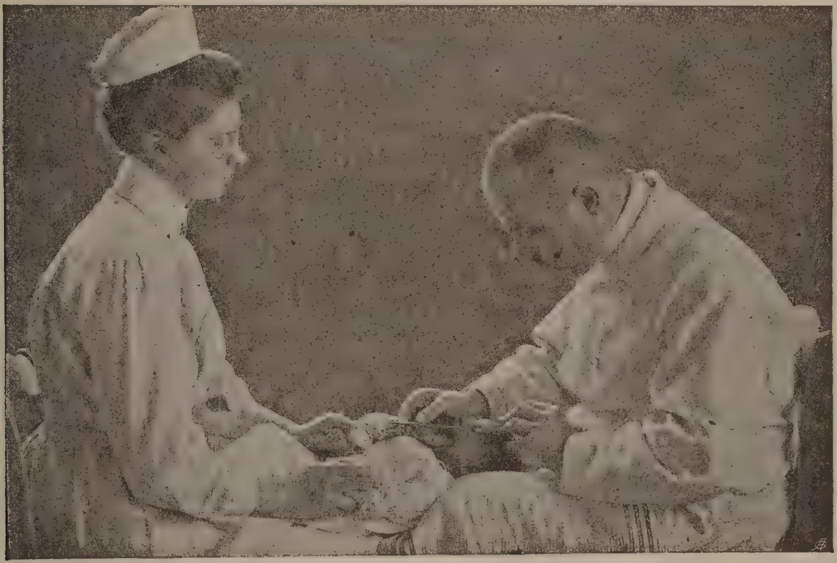

FIG. 13.—Examination in Blennorrhœa Neonatorum.

absorbent cotton is taken from a solution of bichloride of mercury, squeezed out with the fingers and laid over the palpebral fissure, the lids are drawn apart, the physician using both hands, and the gush of pus is intercepted by the wet cotton. If this precaution is not observed, and if the disease is in its early stage, when the lids are tense and tightly closed by a spasmodic contraction of the orbicularis, it may easily happen that when the lids are separated the pus will spout from the eye and endanger other eyes.

Although the greater part of the pus has now escaped little can be seen as yet of the eye, as it needs further cleansing by squeezing a piece of absorbent cotton saturated with a solution of boric acid, or with a physiological salt solution, over it, so that the gentle current of fluid will wash away the remaining pus.

Even now the eye cannot be seen, if the inflammatory swelling is great, without recourse to the lid elevator. This instrument is anointed with vaseline, in order that the mechanical irritation of the eye may be as slight as possible, and then introduced beneath the upper lid. As soon as the lids have been separated by this means new masses of pus come into view from the transition fold. These need to be washed away and then the conjunctiva can be seen to be very red and swollen.

All this is done not only for the purpose of removing the pus, but also to enable us to ascertain the condition of the cornea. Focal illumination from a lamp situated to one side is very well suited for this purpose. The cornea may be clear and bright in one eye, while in the other an oval ulcer with cloudy gray margins and base may perhaps be seen just below its centre, as soon as the pus has been washed away. These ominous corneal ulcers that occur in blennorrhœa commence in the same way as other ulcers of the cornea. The surface is at first dull and presents a slight diffuse cloudiness; the opacity increases; a purulent infiltration develops; the epithelium is broken off and the parenchyma breaks down. Such an ulcer of the cornea is a serious complication. The gonococci from it may, after perforation, infect the eyeball and penetrate into the iris. They have been traced by *Freund* even to the equator of the globe. But other pathogenic agents take part in the development of the corneal ulcers that are formed during the course of a gonorrhœa. The prevailing theory, based on the findings of *Coppel*, is that secondary infections originate from the toxic products of the gonococci after the corneal epithelium has been injured.

II.—The Pathogenic Agent of Conjunctivitis Gonorrhoica

A number of smears should be made of the pus on slides and stained. For diagnostic purposes it is necessary that, in addition to those stained by the ordinary methods, a control preparation should be made according to *Gram's* method, by which the gonococcus is decolorized very promptly. Examination of the slides will then reveal the diplococci, stained red by *Gram*, occupying by preference the pus cells, but also lying near and between them. The peculiar, kidney-shaped contour of the cocci should be noticed particularly.

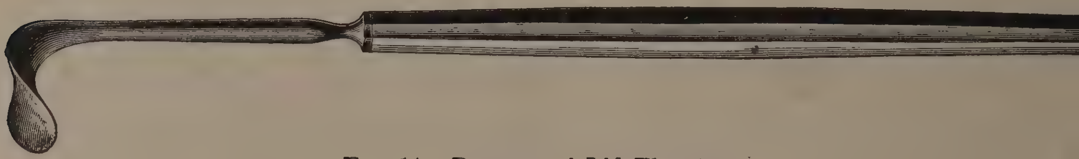

Fig. 14.—Desmarres' Lid Elevator.

Cultures grow best in media which contain 50% of uncoagulated human serum. When a large quantity of pus is used to impregnate ordinary media gonococci can frequently be cultivated in them, but usually the attempt to cultivate gonococci in simple media is unsuccessful. The colonies formed in serum agar are very delicate, when serum bouillon is used a mucous sediment is deposited. It is an interesting fact that gonococci affect man alone; when they are introduced into the bodies of other animals they quickly die and cause circumscribed inflammations only through their endotoxic action. Inoculations upon the mucous membranes of animals have always proved unsuccessful, while a living pure culture applied to the human urethra always excites a typical gonorrhœa and is always highly pathogenic to the mucous membrane of the human eye.

III.—The Course and Prognosis of Conjunctivitis Gonorrhoica

The first signs of this disease are usually noticed between the second and the fourth day after birth. Ordinarily both eyes are involved from the start, sometimes the inflammation appears first in one eye and later in the other, in rare cases one eye alone is affected.

The immediate cause of the disease is the entrance of secretion containing gonococci into the conjunctival sac. While the head of the child is passing through the genital passages of the mother, during labor, the lids may become separated and permit the entrance of an infectious secretion from the mucous membrane of the vagina. The danger that this may happen is increased by a premature rupture of the membranes and by a prolonged labor. Many cases have been reported in which the children have been born with active blennorrhœa and with corneæ already ulcerated or perforated. In most of these cases the labor was complicated by abnormal positions of the child, premature rupture of the membranes, or delay, with frequent manual examinations.

But the infection may take place after birth from secretion adherent to the lids, which is brought into contact with the conjunctiva by an improper method of cleansing. It also sometimes happens that the infectious secretion comes not from the mother, but is conveyed to the child from some third person. Occasionally also a mother infects a child as the result of her uncleanliness as regards her lochial secretion, which contains gonococci. In all such cases we have to deal with a late infection. However the infection may take place the disease commences with characteristic symptoms and its course may be divided into three periods.

The first stage is that of infiltration. The lids swell rapidly so that the eye can no longer be opened. At first this swelling is so tense that it is difficult to separate the lids, even with the aid of an elevator. The palpebral conjunctiva appears bright red as far back as the transition fold; sometimes the bulbar conjunctiva becomes red and chemotic, a condition that indicates the presence of a malignant infection and renders the prognosis grave. The entire mucous membrane still looks smooth and lustrous. A watery secretion, frequently having a yellowish tinge, exudes from the palpebral fissure and indicates the increased secretory activity of the conjunctiva.

This stage lasts only a few days. The first sign that it has passed is the change in the nature of the secretion into thick, yellow pus. The designation, stage of the pyorrhœa, has been aptly applied to this, the second stage, for the pus pours in streams from the eyes and is constantly renewed. A few minutes after such an eye has been cleansed a great quantity of pus is present again. This stage lasts for weeks, during which there are fluctuations in the quantity of pus secreted.

The tense swelling of the lids decreases gradually, folds reappear in their skin and their mobility improves. Meanwhile the appearance of the conjunctiva itself undergoes notable changes. As soon as suppuration begins the mucous membrane becomes more darkly red and loses its smoothness. The palpebral conjunctiva, as far as the transition fold, gradually assumes a velvety appearance, because the papillæ have begun to hypertrophy. These symptoms indicate that the disease is about to enter its final stage. As the secretion gradually loses its purulency, becoming muco-purulent and finally catarrhal, the papillæ become greatly enlarged until the conjunctiva looks as if it were cut to pieces and is covered with ragged elevations. This stage of papillary hypertrophy may persist for two months, or even longer, but it finally undergoes involution and leaves the mucous membrane once more pale and smooth. The conjunctiva may recover completely from a gonorrhœal conjunctivitis without having suffered permanent damage, and this is the more remarkable because, throughout the course of the disease, it has passed through important pathological changes. In the first stage the œdematous swelling, the great distention of the vessels and the infiltration of the tissue predominate. The epithelium of the palpebral conjunctiva is loosened and even absent in places. In the third stage the most prominent symptom is the enormous hypertrophy of the papillæ with large vessels in their connective tissue stroma. But the tarsus is exempt throughout the entire course of the inflammation, a fact that goes far to explain why no serious cicatricial changes persist after its subsidence.

Unfortunately the course of the disease is very often complicated by involvement of the cornea. When this happens it is a rare occurrence to have a simple infiltration form and retrogress completely; usually the infection penetrates into the cornea and leads to the formation of an ulcer in one of two places.

Sometimes a yellowish gray infiltrate appears at the margin of the cornea, soon becomes confluent and forms a crescentic ulcer parallel to the limbus, like that met with in catarrhal inflammation, and usually this extends until it has circumscribed the entire periphery and caused the whole cornea to break down into pus.

A type much more frequently met with is one in which a slight, smoky opacity appears a little below the centre of the cornea, increases until the epithelium is thrown off and a horizontally oval ulcer is formed, which exhibits an unfortunate tendency to penetrate into the parenchyma. The situation of this ulcer is characteristic. It corresponds to the portion of the cornea which, when the eyes are closed and turned upward, lies directly beneath the palpebral fissure. This localization is due to the fact that the pus is present in greatest abundance within the palpebral fissure and so enables the toxic products of the microorganisms to produce their most virulent effect upon the cornea at this place. The course of this ulcer differs from that of the other. The result may be termed excellent when it heals without perforation and cicatrizes with the aid of an extensive vascularization extending to it from the margin of the cornea. A leucoma is left which may clear up so much that in later life a delicate macula alone may testify to the gravity of the present situation.

Too often the progress of these ulcers is not checked until they have caused perforation of the cornea, and the final result then depends, among other things, on the size of the ulcer and the way in which the perforation takes place. Most frequently a prolapse of the iris occurs and is productive of a leucoma adherens· or the prolapse may heal in the form of an ectatic cicatrix, which results in a larger or smaller staphyloma. If the ulceration maintains its activity in spite of the perforation the infection penetrates into the eyeball and the final result is phthisis bulbi.

The behavior of the lens at this time deserves special attention. If it moves forward, so as to come in contact with the cornea when the aqueous escapes through a central perforation, the purulent inflammation in the cornea excites an irritation of the epithelium of its anterior capsule. This proliferates and an anterior capsular cataract is produced, which forms in later life an evidence of what has taken place. When the perforation occurs suddenly, particularly if the child is straining hard at the time, the lens may be dislocated and appear between the margins of the lids.

So much concerning the course of the disease as regards the eye itself, but the entire organism not rarely participates. Infants with this disease may have a gonorrhœal stomatitis, which usually runs a benign and rapid course and is frequently overlooked. It may be caused by transference of secretion to the mouth by the hand of the child, but the gonococci may also reach the throat and mouth by way of the lachrymal passages.

The connection between the ocular inflammation and the inflammations of joints met with in infants is more important. It is evident that some of the gonococci may enter the blood and be deposited metastatically in the joints. The presence of gonococci in intra- and periarticular exudates has been demonstrated by several authors, particularly by Deutschmann, Lindemann and Koch. These arthritic inflammations appear usually in the third or fourth week of the disease, but may present themselves sooner. The knee, wrist, ankle and sometimes the elbow, are the joints ordinarily affected. The course is febrile, but as a rule benign, and periarticular abscesses are rarely met with.

It is evident from what has been said that conjunctivitis gonorrhoica neonatorum is a very serious disease. Not long ago, and in some countries even at the present day, the majority of the persons living in asylums for the blind owed their incurable blindness to blennorrhœa neonatorum. Statistics vary and details are burdensome, so a few examples will suffice to impress this fact. Magnus found that of 1,046 persons who became blind during the first year of their lives, 753 lost their sight through blennorrhœa. In some asylums 30, 35, and even 60% of the inmates had been rendered blind by this disease. Fuchs estimates that the number of the blind in Europe amounts to 300,000, and that more than a tenth of these owe their condition to blennorrhœa neonatorum. Hence no case of this disease should be esteemed as not very serious. The treatment of every case should be transferred to the specialist as quickly as possible, but the general practitioner should be thoroughly conversant with the treatment, in the first place because it is not always possible to transfer a patient to the specialist's care, and in the second place because there is an intimate connection between the treatment and the prophylaxis of this disease, the latter of which offers a fruitful field for his work.

13. Treatment and Prophylaxis of Conjunctivitis Gonorrhoica Neonatorum

A. Therapy of Conjunctivitis Gonorrhoica

The fundamental principles of the treatment of blennorrhœa are substantially the same as those of the treatment of other acute infectious inflammations of the conjunctiva, but they must be applied more rigorously. because the eye is in greater danger in a gonorrhœal infection than in any other conjunctival disease yet discussed.

As soon as a child with blennorrhœa is admitted to the clinic it is isolated and confided night and day to the care of a special nurse. The nurse must first of all be familiar with the care, cleaning and feeding of children; for this reason it is well when the mother can be admitted with the child. The nurse must know that she is dealing with a dangerous disease, and that, for the protection of her own eyes, she must cleanse and disinfect her hands with painstaking care each time after she has had occasion to touch the child. Her duties are very fatiguing and perilous. During the absence of the physician her instructions are to cleanse the eyes in the manner about to be described.

It is of fundamental importance in cleansing the eye that the cornea must not be touched either by whatever is used as a sponge, or by the hand. For this reason the secretion coming from the palpebral fissure should always be wiped away by a movement of the hand from the temporal toward the nasal side. The lower lid is to be drawn down and out from the eyeball and the conjunctival sac may then be thoroughly irrigated.

Although such lotions as solutions of boric acid in the strength of 1:300, of bichloride of mercury, 1:5,000 or 1:10,000, of permanganate of potassium, 1:5,000, and of oxycyanate of mercury 1:2,000 are useful and harmless in other forms of conjunctivitis, it is doubtful if their use in blennorrhœa is advantageous in any way and I have discarded them for the following reasons:

It has never been demonstrated that the gonococci in the infected conjunctival sac can be destroyed more quickly by their aid. The bactericide power of solutions of the given strength is exceedingly minute under conditions in which the cocci are partly in the tissue and partly in the folds of the conjunctiva, conditions that are in no way comparable with those present in a test-tube experiment. When the cleansing of one eye is performed with one of these solutions and that of the other with ordinary salt and water, the gonococci decrease no more rapidly and no sooner in one eye than in the other. But even if a slight antiseptic action might occasionally be produced, such an advantage would be overbalanced by the injuries inflicted.

It must never be forgotten that if an eye is to be kept as free as possible from the infectious secretion during the stage of pyorrhœa, it must be cleansed every 15 or 30 minutes. It is astonishing in how few minutes after such an eye has been cleansed the pus will reappear. If a normal eye is irrigated in this way with one of these antiseptic solutions for a single day a noticeable irritation will be produced, even though different solutions are employed in turn. In an eye with such an inflammation as this we are now dealing with, in which the tissues are extremely sensitive and loose, this irritation is followed by injury to the epithelium, for the antiseptics are poisons not only to the protoplasm of bacteria, but also to the protoplasm of cells. This injury might be accepted as of not serious importance if it were counterbalanced by decided benefit in other ways, but clinical experience does not show this to be the case.

I have likewise discarded the application of ice-cloths. The erroneous theory has prevailed that the infectious process could be influenced by the application of cold, but thermometric tests have proved that the cooling of the lid thus produced does not extend to the conjunctival sac, and if pieces of ice wrapped in cloth are applied, the thin, tense and badly nourished lid skin itself may necrose. Another reason why this kind of application should be discontinued is that even the most conscientious nurse will get tired; she believes that she has done her duty when she has placed the cloth upon the eye, and there she lets it lie.

Stagnation of the pus must be prevented, and this can be done only by regular cleansing and irrigation of the eye. The fluid used for irrigation should be slightly warm. There is little harmony between ice-cloths on the one side and warm irrigation on the other. Warnings have frequently been uttered that when such an antiseptic as oxycyanate of mercury is used the irrigation must be performed in such a manner that the fluid cannot come into contact with the cornea. Do all nurses, or even all physicians, know this? Procedures that are theoretically curative, but are incapable of being carried out practically, should never be recommended.

So far as the nurse is concerned in the treatment, she has only to maintain the constant cleansing of the eye, the physician himself attends to the rest. The classical method of treatment in this disease is with silver nitrate. At first, when the swelling of the lids is so tense that the conjunctiva can be reached only with considerable difficulty, little can be done in addition to the irrigation, except to watch the course of the disease. After a few days, when the tense swelling has relaxed, the surface of the conjunctiva has lost its smoothness, the secretion has become purulent and the papillæ are beginning to enlarge, the lids may be carefully everted, and a 1 or 2% solution of silver nitrate may be painted over the surface of the palpebral conjunctiva in the same way as in adults and in other forms of conjunctival disease. One eye is treated first, then the other. Nitrate of silver in solid form should never be used. These applications may be made at average intervals of twenty-four hours, and for a short time thereafter the eyes may be covered with moist compresses to mitigate the reaction, the irrigations to be then resumed. Under this treatment the swelling and secretion gradually subside, in the course of 4 or 5 weeks in grave cases, during which time the secretion becomes thinner, muco-purulent and finally scanty.

Thus far the course followed has been that of a normal case. When corneal complications occur we must confess our impotency, if we are frank. The advocate of the use of ice-cloths immediately discontinues them, but this does not help those who do not use them at all. I agree with the majority of ophthalmologists in the view that even when corneal complications are present treatment with silver nitrate is still permissible, because everything depends on controlling the conjunctival disease as quickly as possible. When the infiltrates in the cornea are still superficial atropin is employed to control the hyperæmia of the iris, or the iritis. When the ulcer has perforated and a prolapse of the iris is present the latter should be left quiet as long as possible. Experience has taught us that perforation favors the healing of the ulcer itself and no attempt should be made to excise, or to cauterize, the prolapse until the disease has run its course.

Substitutes for silver nitrate in the treatment of this obstinate disease, which lasts a long time under the best conditions, have naturally been sought for and employed, but rarely have any of them proved its equal, none have ever proved superior.

The question arises what does this therapy accomplish? Some ophthalmologists lay special stress upon this or that material for irrigation and get good results without the use of silver at all; others have returned to the classical treatment after a brief abandonment in favor of a substitute; others again advocate the new remedies; each can exhibit good results, but each has his mishaps. It seems hardly proper for a scientific investigator to speak dogmatically in this regard. Still we continually find, printed in text-books and presented in lectures, the statement that by timely treatment the cornea can be saved with certainty. That is simply untrue, as every objective investigator must admit when he examines the records of any one clinic for a period of ten or twenty years. Such an examination will reveal how one modification after another has been tried in the treatment of blennorrhœa, and how corneal complications have occurred under all forms of treatment, even when the cases have been under observation from the very beginning. This shows that the fate of the infected eye depends not alone on the treatment, but also on the virulence of the pathogenic agent and on the susceptibility of the patient. In mild cases it is easy to save the cornea, in grave cases this is not always to be accomplished so readily. *I am convinced that in blennorrhœa it is a matter of indifference what known remedy we use in addition to the cleansing of the eye, the infection takes its legitimate course.* My estimation of the efficiency of our therapy is at variance with that of not a few ophthalmologists.

The feature that has hitherto characterized all experiments and therapeutic propositions in conjunctivitis gonorrhoica is the attempt to find a means to kill the gonococci as thoroughly and quickly as possible. This idea has floated before the minds of most, if not of all, of those who have recommended chemically acting remedies for the treatment of blennorrhœa. But there have not been lacking others who expected nothing from such means, because they were convinced that no bactericide has the power to attain the desired result, and confined their efforts to irrigation of the eyes. When, without prejudice, we view the conditions present in gonococcal infection of the conjunctiva we see reproduced on this membrane the same spectacle with which we are familiar in other organic infections. Our antiseptics are unable to disinfect this mucous membrane. Gonococci are still present when a blennorrhœa has been treated for weeks with silver, or other remedies, and they are still present, a point worthy of notice, after the organism has got the better of the infection, after the disease has been overcome and they can no longer do harm in the eye. Where is the cure by means of antiseptics?

In the future I believe that the treatment of blennorrhœa will be conducted along different lines. It does not happen very often that the pathogenic agent is directly and quickly killed when we overcome an infection anywhere in the body; it suffices perfectly when the pathogenic power, the virulence of the agent, is paralyzed in the affected person. As soon as this is attained the organism is ready to deal with the infection entirely by itself, in the very best manner. We constantly meet with this biological fact as we follow a spontaneous recovery from infectious diseases. Diphtheria bacilli may be obtained from a conjunctiva for a considerable period of time after a diphtheritic conjunctivitis has run its course, but they are harmless to the patient because the organism has become immune. Our knowledge concerning the conditions that obtain in the spontaneous recovery from gonococcal infection of the eye is slight as yet, but gonorrhœa differs from diphtheria in that a general immunity of the entire body is either not formed at all, or is not demonstrable.

There is also a difference between urethral gonorrhœa and gonococcal infection of the eye in that a chronic gonorrhœa of the urethra is not uncommon, while a chronic gonorrhœa of the conjunctiva is unknown. In the eye the gonococci generally decrease gradually along with the subsidence of the disease, but it has been shown by accurate investigation that they outlast the suppuration. Thus *Groenouw* found gonococci in the eye for twenty-five days after the cessation of the secretion, and, even though they do not always persist as long as this, it is certain that, though present, they no longer cause injury during the final stage of the disease. It must also be remembered that conjunctivitis gonorrhoica may recover spontaneously without any treatment, though more frequently not until irreparable injury has been done to the cornea.

This condition of tolerance is attained by the conjunctiva under the influence of the specific infection and we may conceive of it as a kind of local immunity.

The final aim of all our therapeutic efforts should be to bring about as quickly as possible, without danger to the eye, such a local insensibility of the conjunctiva to the gonococcal infection, but we are still far remote from such an ideal, because we are ignorant of the processes that take place in the cells of the conjunctiva. We know that such a local specific immunity always develops slowly and only under the influence of the specific infection, and that during this development serious injuries to the cornea frequently occur. The thought suggests itself that perhaps this immunity might be attained at the outbreak of the disease by means of active immunization with dead cultures of gonococci. In adults this might possibly be done, in a manner analogous to the active immunization to typhoid fever and cholera, but should dead gonococci be injected under the conjunctiva, or into the muscles of an infant suffering from blennorrhœa when we know that a general immunity to this pathogenic agent is unattainable, if it even exists? It seems more rational to adopt passive rather than active immunization in gonococcal conjunctivitis. In this form of serum therapy it is the purpose to counteract the infectiousness of the gonococci and to lessen their virulence by preventing the development of new generations, which act aggressively, during the course of the infection. If such a result can be obtained it will furnish the preliminary condition for facilitating a spontaneous cure. The gonococci need not be killed; with such aid as this the organism can perhaps deal with them as it does with other morbific agents by the aid of serum therapy. The interesting clinical problem is thus presented to ascertain whether, and to what degree, such a result can be attained by means of serum therapy in blennorrhœa. It is evident that it can be done only by means of a specific serum. This therapeutic proposal is new. Some experiments have been made with normal serum, but this is unsuited for the purpose. I have therefore adopted in the treatment of blennorrhœa a specific gonococcus serum that has been prepared by Professor *Ruppel*, in Hoechst. This serum is used in the form of a dry, fine powder and is dusted into the conjunctival sac after each irrigation. It quickly becomes dissolved in the fluid and the secretion of the conjunctiva, so that the gonococci are thus brought in contact with a great quantity of the specific protective substance. The irrigations of the conjunctival sac may be made also with a 0.85% solution of sodium chloride to which sufficient serum has been added to make a concentration of 1:100. In this way the attempt may be made to act directly upon the gonococci through local contact with the specific antibodies. No resorption of the antibodies from the conjunctival sac takes place and the organism cannot be immunized by passive immunization through the conjunctiva.

These thoughts are propounded that it may be clear why I combine with the classical silver treatment this form of serum therapy. The critical observer cannot remain satisfied with the treatment of blennorrhœa as it has hitherto been practised, new ways must at least be tried, and it seems rational to seek local aid from the specific protective material of the blood serum in this infectious disease. Whether the results of this combined treatment will prove to be better, or not, cannot be told as yet, but at all events the application of the dry pulverized serum is harmless. It may be dusted over a corneal ulcer and from this we know that it does not sterilize the infected corneal tissue. The irrigation of the conjunctival sac with salt solution to which the specific protective substance has been added is certainly less dangerous than the prolonged use of antiseptics that not only do not sterilize the tissues, but are actually harmful to the conjunctiva. But we have not attained the ideal in the treatment of blennorrhœa with this serum alone, as has been proven by observations instituted at my request in Sattler's clinic at Leipsic, and it remains for the future to develop an efficient treatment for conjunctivitis gonorrhoica.

To recapitulate the treatment of this disease: We irrigate the conjunctival sac with salt solution containing serum, then paint the conjunctiva with a 1% solution of silver nitrate, allow the reaction from this to subside for an hour or two, and then dust in the powdered serum after each irrigation. The application of the silver to the second eye will convince any one that it is not made as easily as to the eye of an adult, but the physician must learn how to do it.

B. Prophylaxis of Blennorrhœa Neonatorum

In the prophylaxis of this disease we come to a subject of much interest to every general practitioner. An infectious disease can always be prevented better than it can be cured. This is the case with blennorrhœa neonatorum. Although gynæcologists had striven for a long time to guard the eyes of the infant against infection by appropriate treatment applied to the genital passages of the mother, and although *Kehrer*, *Bischoff*, *Schmidt-Rimpler* and others had tried to prevent the outbreak of the disease by prophylactic treatment of the eyes themselves, yet it remained for *Crede* to win everlasting renown by the discovery of an efficient means of prophylaxis.

Crede's own directions were: "As soon as the umbilical cord has been cut the child is to be freed from the vernix caseosa, with the adherent blood and mucus, in the usual way and then placed in a bath. The eyes are to be washed with a clean cloth, or better absorbent cotton, not with the water of the bath, but with other clean ordinary water. Then, before the baby is dressed, each eye should be opened a little by means of two fingers and a single drop of a 2% solution of silver nitrate brought in contact with the cornea and allowed to drop upon it. No further attention is to be paid to the eye. The instillation must not be repeated if, during the next twenty-four or thirty-six hours, there should be a slight redness and swelling of the lids with a mucous secretion." It would certainly be more judicious to instill the drop of silver nitrate upon the palpebral conjunctiva at the inner canthus than directly upon the cornea.

The results produced by this prophylactic measure were extraordinarily successful. In *Crede's* clinic the percentage of blennorrhœa sank from 10% to 0.2%, and the reports from many other clinics bore testimony to its efficiency.

These brilliant results were due to the power possessed by the silver salt to either kill outright the few gonococci in the conjunctival sac, or to render their colonization impossible. This exemplifies how entirely different is the prophylactic action of silver nitrate from its therapeutic action when the disease has become fully established. In the one case a single drop suffices, in the other the conjunctiva may be painted with it for weeks without destroying the germs which are firmly bedded in the tissue. That we should hold it in high estimation for prophylaxis and think poorly of it as a therapeutic agent when the infection is accomplished involves no contradiction of any biologic law.

Very soon objections began to be made to *Crede's* method. The silver nitrate may excite in the delicate conjunctiva of an infant a sharp reaction characterized by redness, swelling and more or less secretion, as has been demonstrated by *Kuestner*, *Keilmann*, *Cramer* and others. This catarrh may be aseptic at first, but after a few days the ordinary bacteria of the conjunctiva, xerosis bacilli and staphylococci, will be found proliferating therein. Such observations led to many attempts to substitute for silver nitrate other means, of which the best known is protargol; but, in spite of what might be called the naturally necessary inconveniences that attend its use, most authors adhere to its employment, and I myself favor at least a warm 1% solution. In any case the objections made to *Crede's* method are not to the point. Even when a catarrh is caused by the instillation of a drop of a 1% solution of silver nitrate, it does no harm, but soon passes away and we would gladly produce ten such catarrhs if we might thereby prevent a single case of gonococcal infection. But, as a matter of fact, such severe catarrhal reactions are by no means so frequent as has been assumed. We must not forget also that when silver is not used infants are subject to catarrhs caused by other pathogenic agents, in the treatment of which silver is again needed. The overwhelming majority of ophthalmologists agree that the objections which have been made to *Crede's* procedure are weak and that the practice of this most excellent prophylaxis cannot be urged too strongly. But the employment of *Crede's* method in lying-in hospitals alone does not suffice for the prophylaxis of blennorrhœa, as is shown by the fact that too large a number of cases come for treatment from general practice. It has been tried in many states to bring *Crede's* method into general use among midwives, but unanimity has not yet been attained, and the directions are ever changing in a lamentable manner. Midwives should at least be thoroughly instructed in the nature and dangers of the diseases met with in the eyes of infants, and it should be energetically enforced that a midwife call in the aid of a physician as soon as this disease has made its appearance.

It may seem pedantic to describe how a drop should be instilled into the eye, but experience will convince any one that even this little manœuvre requires practice. It is a good idea for students to practise instillation of such a drop into the eyes of each other. Lay the head of the patient somewhat backward and bid him look up; then draw the lower lid away a little from the eyeball and instill the drop gently *without touching the lids with the pipette*, at the same time holding a small piece of absorbent cotton on the skin of the lid with the hand used to draw it out, to absorb and remove whatever overflows, so that the silver salt may not come in contact with the clothing. Likewise it is well to have the patients who need to be treated with drops practise the instillation in the same manner. Children struggle less against the application when the solution is slightly warm.

14. Conjunctivitis Gonorrhoica in Adults and the Protective Dressing for the Uninfected Eye

Yesterday evening the eyes of this patient appeared to be perfectly well. This morning one of them is the seat of a grave disease. Both lids of the affected eye are enormously swollen, tense and red, and the patient can no longer open them. A thin, yellowish reddish fluid resembling serum wells forth from between the lids in considerable quantity. After the lids have been carefully separated by means of the elevator the conjunctiva is seen to be of a deep red color and swollen. The conjunctiva bulbi is highly chemotic and surrounds the cornea with a firm, doughy wall, overlying it in some places. Large and small hemorrhages may be seen in almost all sections of the scleral conjunctiva. The upper lid cannot be everted to bring the upper transition fold into view; the lower transition fold springs forward in a thick fold, on which, as well as on the conjunctiva of the lower lid, may be seen gray membranes, or at least gray places. The cornea is still intact and the other eye is still well.

The patient complains not only of burning, but of severe pain in the inflamed eye. The gentlest touch upon the bluish red, tense lids causes exquisite pain. The patient has a feeling of chilliness and his temperature is above normal. The preauricular lymphatic gland is swollen. An acute conjunctivitis that has reached such a height after an incubation period of only a few hours excites at once the suspicion that it has been produced by an infection with gonococci. The presence of these microorganisms is revealed by a microscopic examination of smears made from the conjunctival secretion, while the history and personal examination of the patient is apt to reveal the presence of a urethral gonorrhœa.

Ordinarily this disease is communicated to the eye by the finger of the patient himself, but it may be carried by washcloths, handkerchiefs and the like—a monition that a person suffering from gonorrhœa should be very careful lest he infect his eyes in this manner. It is certainly amazing that this disease of the eye is not more frequently met with in such patients, because any itching of the eye gives rise to an impulse to rub or wipe it. Many cases have been described in which the infection was produced through a filthy custom prevalent in some localities, that of bathing their eyes in urine as a sanative measure, but when the urine happens to contain gonococci the measure proves to be the opposite of sanative.

Autoinfection is not the only way in which this disease is generated; many an eye has been infected from the gonorrhœal secretion of the urethra of another person.

Although gonorrhœal conjunctivitis is a very serious disease in an infant it is far more grave in an adult, because in the latter the cornea is endangered to a much higher degree. The disease in adults differs from the purulent ophthalmia of infants not only in its more tempestuous development, but in the more marked involvement of the conjunctiva bulbi, which forms an infiltrated and chemotic, overhanging wall about the periphery of the cornea, so that the infectious secretion stagnates behind it and erodes the corneal tissue. At the same time the great infiltration of the conjunctiva compromises directly the access of nutriment to the cornea through the marginal network of vessels, so that the statement is justified that the fate of the cornea in this disease depends greatly on the condition of the conjunctiva. Because of this danger the general practitioner is accustomed to turn over such patients at once to the care of the specialist.

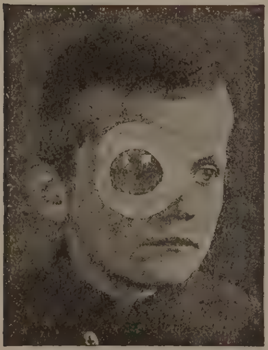

FIG. 15.—*Buller's* Shield.

But he has a second task to perform. As a rule only one eye is infected at first, and therefore measures must be taken immediately to guard the other eye and prevent its subsequent infection. This is accomplished by the application of a suitable protective dressing, which may be made in several ways. Some ophthalmologists content themselves with a pad of cotton secured all around by means of adhesive plaster or collodion (*Fuchs*, *Knies*). Others, like *Paulsen*, use gummed paper, which is moistened at the edges with chloroform to dissolve the gum and make it adhere. A method which is preferable to these is to build up the uneven margin of the orbit with cotton, cover the eye with a large watch crystal and lute the margins of the glass to the subjacent cotton with collodion.[1] *Snellen* has devised a leather cup with an aperture in its centre to contain a plain glass. Others use a transparent celluloid capsule. Such a dressing must absolutely exclude any possibility that the secretion from the diseased eye may enter the healthy one either during sleep, or when the head is held in any possible position. This dressing must be changed from time to time because otherwise the eye will become irritated, even without infection, as the result of faulty evaporation. The patient must put up with the inconvenience it occasions. After the general practitioner has applied such a protective dressing to the healthy eye and turned over the patient to the specialist he has done his duty.

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[1] This dressing is generally known in this country as Buller's Shield, as the late Dr. Buller of Montreal, Canada, is credited with having devised it.

The treatment is conducted along the same lines as that of blennorrhœa neonatorum. This disease passes through the same stages as the other. After a few days the inflammation reaches its height, the thin fluid secretion becomes purulent and the pus wells constantly from the palpebral fissure. The tense swelling of the lids goes down, folds reappear on the skin, the tension of the conjunctiva bulbi relaxes and the conjunctiva palpebrarum exhibits in the transition folds an increasing proliferation of the papillæ. In auspicious cases the secretion diminishes for some weeks until it finally ceases. The papillary thickening of the conjunctiva persists considerably longer and the entire course of the disease requires several weeks.

During the first stage the secretion should be constantly removed by irrigations of the conjunctival sac with warm 0.85% salt solution to which gonococcus serum has been added. Other ophthalmologists employ such solutions as they prefer. In these irrigations I do not use the great quantities of fluid that many ophthalmologists allow to run over the eye. Beyond all question the conjunctiva and cornea must react to such treatment. All of the gonococci cannot be removed with the secretion and any injury to the epithelium must be avoided. It is particularly necessary to wash away carefully the masses of secretion that lie beneath the chemotic conjunctiva and to do this without injury to the epithelium. As soon as the swelling of the infiltrated lids goes down and the conjunctiva looks folded and uneven on account of the hypertrophy of the papillæ, a 1% solution of silver nitrate should be applied to the palpebral conjunctiva. This may be combined with the local application of the specific gonococcus serum. When corneal complications occur we are fairly powerless, the same as those who otherwise are accustomed to pride themselves on their therapy. Later the sequelæ of these complications, staphylomata and leucomata, may require operative treatment.

We have still to consider what are the most frequent corneal complications, when we have to fear them and what is the prognosis of the disease. Sometimes the cornea becomes involved during the first stage, but usually not until the period of suppuration. As a rule the complication takes the form of an ulcer either at the margin, or in the central portion of the cornea, which goes on to perforation in both situations. But in very severe cases, in which the cornea receives insufficient nourishment on account of the extreme chemosis, the entire cornea may simply disintegrate without ulceration.

The prognosis of the gonorrhœal conjunctivitis of adults is always to be considered as very grave, but it is not easy to predict the final result because very serious cases may recover completely. As a rule the involvement of the conjunctiva bulbi furnishes some points to assist us. If this is from the first highly chemotic and infiltrated we must expect the worst. In such desperate cases we may scarify this conjunctiva with a knife in order to disburden it. But if the inflammation is confined more to the palpebral conjunctiva and the transition folds the prognosis is more favorable. Membranous deposits on the conjunctiva excite apprehension because the rigidity of the tissue is indicative of a virulent infection. But the final result is uncertain; the disease is extremely serious and we are unable to master it therapeutically.

Metastatic Conjunctivitis Gonorrhoica.—Persons suffering from gonorrhœa sometimes develop another and benign form of conjunctivitis gonorrhoica which is produced by metastasis. It is well known that arthritic inflammations take place during the course of a urethral gonorrhœa and that other organs, like the heart and lungs, likewise suffer in this way. In ophthalmology also we meet with endogenous gonococcal diseases, such as gonorrhœal iritis and the form of gonorrhœal conjunctivitis now under consideration. Cases of this nature have long been known to dermatologists, have been described many times in ophthalmological literature and are occasionally met with. An acute conjunctivitis appears suddenly during the course of a urethral gonorrhœa, but the secretion contains no gonococci, or other germs. Sometimes it exhibits a certain rhythm, the conjunctivitis returns with each recurrence of the gonorrhœa. A characteristic feature of this form of metastatic inflammation is that the pathological redness is confined rather to the transition folds than to the palpebral conjunctiva. The disease is benign; the only treatment needed is bathing of the eye and rest in bed.

15. Catarrh of Newly Born Infants not of Gonorrhoeal Origin and the Significance of the *Gram* Negative Diplococci of the Conjunctiva

The worst need not always be anticipated when an infant has a purulent conjunctivitis, for modern research has established the important fact that gonococci are absent in many cases that present the clinical picture of blennorrhœa neonatorum. Gonococci were found in only 63 out of 92 cases examined by *Kroner*, in 64 out of 103 by *Widmark*, in 14 out of 40 by *Groenouw* and in only 70 out of 165 by *Morax*. Hence, the occurrence in infants of such non-gonorrhœal catarrhs is not so very rare. More important than these statistics is the question by what morbific agents these non-gonorrhœal catarrhs are produced.

The first group comprises all possible kinds. There have been found in cases that could not be differentiated clinically from true gonorrhœal inflammation of the eyes—1, the pneumococcus by *Axenfeld*, *Gifford*, *von Ammon* and others; 2, the staphylococcus pyogenes aureus; 3, the *Koch-Weeks'* bacillus by *Morax* and *Gonin;* 4, *Friedlænder's* pneumonia bacillus by *von Ammon;* 5, the bacterium coli by *Axenfeld* and *Bietti;* 6, the diplobacillus of *Morax-Axenfeld;* 7, the streptococcus pyogenes; 8, the influenza bacillus; 9, the diphtheria bacillus. In this group may also be included the cases, that are by no means rare, in which no bacteria were found, except the ever present xerosis bacillus, although the clinical pictures were typical of blennorrhœa. Probably this form of blennorrhœa without positive bacterial infection is due to a traumatism inflicted upon the lids and conjunctiva during parturition, which enables a parasite harmless in itself to excite a vigorous secretion. Premature and feeble children are prone to catarrhs of the eyes. *Crede's* instillation of silver nitrate occasionally excites irritation and a purulent secretion that lasts for several days. The cause of catarrhs with no characteristic findings must be explained in some such way. The pathogenic agents in this first group are so characteristic that the etiology may be easily and quickly determined.

The whole subject is rendered more complex by the fact that in such inflammations still other microorganisms are met with on the conjunctiva of infants, which closely resemble gonococci and have been termed pseudogonococci. First among these is the micrococcus catarrhalis, which belongs to a group of *Gram* negative diplococci that resemble gonococci, and is met with frequently in the nasal cavity and in bronchitis. It differs from the gonococcus in often being somewhat larger and in appearing not to fill the cells so full when examined under the microscope. These characteristics are not distinctive, but, in contrast to the gonococcus, the micrococcus catarrhalis grows in all media and even below 20°. The cultures differentiate themselves in various ways from those of gonococci, and it is the most resistent of all in the group of *Gram* negative diplococci.

The differentiation of the meningococcus intracellularis *Weichselbaum* from the gonococcus is much more difficult. It is frequently larger, is not always so reniform and it grows somewhat more vigorously in blood serum; but an agglutination experiment is necessary to make a differential diagnosis. A high value meningococcus serum agglutinates meningococci, but not gonococci.

The diagnosis of a blennorrhœa neonatorum is no longer so simple as it used to be and the question arises: How shall the physician deal with such cases? It is quite evident that such a differential diagnosis, for which cultural methods and laboratory contrivances are necessary, cannot be demanded from the general practitioner, although he should be instructed in the scientific position of ophthalmology. The question should read: How can we to-day in practice make the diagnosis of blennorrhœa?

There are certain clinical differences between gonorrhœal conjunctivitis and the catarrhs of non-gonorrhœal nature. As a rule gonorrhœal conjunctivitis lasts much longer and endangers the cornea much more than the other forms of inflammation, which frequently run a mild and rapid course. But it is not permissible in a scientifically conducted clinic to rely upon the course alone for an etiological diagnosis. The etiological diagnosis must be made as quickly as possible. The disease loses its terror as soon as the true cause is known not to be gonorrhœal. In all such cases the secretion must be examined microscopically, especially after staining according to *Gram's* method.

To interpret these slides correctly it is important to know to what extent and how frequently, as compared with gonococci, the other *Gram* negative diplobacilli occur in the eye. Meningococci are met with in the eye very rarely. They may be found occasionally on the conjunctiva of even a healthy person during an epidemic of meningitis. They were found for the first time on the conjunctiva of a man, who neither had meningitis nor had been in contact with a person suffering from that disease, by *Bruns* in *Axenfeld's* clinic. But, so far as is now known, they play a very evanescent part in blennorrhœa and the catarrhs of newborn children. The conditions are somewhat different regarding the micrococcus catarrhalis, which is met with in the eye more frequently than has been thought. Thus far it has not been found in acute inflammations of the conjunctiva, but rather in the sparse secretion of chronic conjunctivitis, together with xerosis bacilli, staphylococci and diplobacilli. Clinical cases of blennorrhœa in which diplococci resembling gonococci have been found have always been proved by culture to be gonococcal. This indicates that the micrococcus catarrhalis does not play an important part in blennorrhœa. *Abelsdorf* and *Neumann* have met with it only in postoperative conjunctivitis, but in this form of inflammation chemical irritation is present and it is quite possible that the micrococcus catarrhalis proliferates here as a saprophyte. It is also possible that it may occasionally cause a simple catarrh, but whether it is able to produce an infantile catarrh, which may be mistaken for blennorrhœa, is very doubtful; at any rate it has not yet been proven. The cultural test is final, as this germ is cultivated easily, while the gonococcus is not.

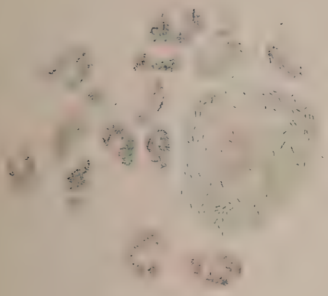
In spite of the difficulties in the way of a differential diagnosis the *Gram* stain furnishes the practical diagnosis of gonorrhœa of the eye *so long as the clinical picture of blennorrhœa is present.* Under certain circumstances this may be of medico-legal importance. It would certainly be well if the aid of cultures could be obtained in all such cases, but cultures easily miscarry for many reasons, while the slide does not miscarry and important decisions may depend upon it. Masses of intracellular, *Gram* negative diplococci associated with the clinical picture of blennorrhœa are in all probability gonococci, but when associated with mild catarrh, they are more likely to be micrococci catarrhales. Of course in such mild catarrhs it is possible that they are gonococci of slight virulence; in such cases the differentiation must be made by culture. There is not the slightest support for the idea that the *Gram* negative diplococci met with on the conjunctiva may have come from gonococci that had become avirulent, or for the idea that gonococci may occasionally develop from other *Gram* negative diplococci; these microorganisms are of different species.

There are certain clinical signs that indicate the non-gonorrhœal nature of this disease in spite of its resemblance to blennorrhœa; it frequently commences later, perhaps between the sixth and twelfth days, the eyes are not affected so simultaneously, the swelling of the lids and the quantity of secretion are not so marked; but such differences as these are insufficient for diagnosis in all cases. An occlusion of the orifice of the naso-lacrymal duct may also cause the development of a purulent conjunctivitis in an infant. The lacrymal fluid, which accumulates in the lacrymal sac in such a case, decomposes after a little while and gives rise to a purulent secretion; therefore the possibility of the existence of such a condition must be considered in every case of infantile catarrh, particularly after the suspicion of a gonococcal infection has been allayed. Either one or both ducts may be occluded. In such cases it does not escape the careful observer that the palpebral conjunctiva is only moderately injected and that the bulbar conjunctiva shows little or no injection. In order to determine whether such a congenital stenosis of the lacrymal duct is present or not, the secretion is washed out of the conjunctival sac and pressure is made on the lacrymal sac. If a quantity of pus issues then from the punctum, the diagnosis of a closure of the lacrymal passage is positive. Usually the only treatment necessary is to teach the parents to massage the lacrymal sac and to press out its contents several times a day; the passage is opened spontaneously in a short time and the purulent catarrh then disappears. The introduction of probes may prove necessary in obstinate cases.

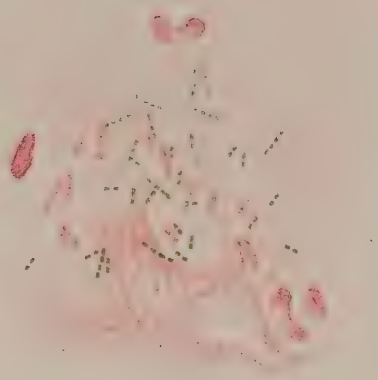
The treatment of other cases, in which the microscopical examination has proved the absence of gonococci, consists simply of irrigations of the conjunctiva performed regularly to prevent stasis of the pus; when the secretion does not diminish rapidly the solution of silver nitrate may be applied. The prognosis of all non-gonorrhœal infantile catarrhs of the eye is very good.

16. Vernal Catarrh

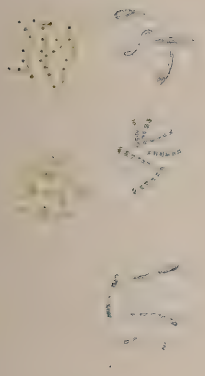
With the advent of spring patients with this disease begin to suffer in a peculiar manner. In February, or more often in March or April, they commence to complain that their eyes are very sensitive to light, heat, wind and weather, and, as the sun's heat becomes greater, they have a peculiar itching of the eyes.



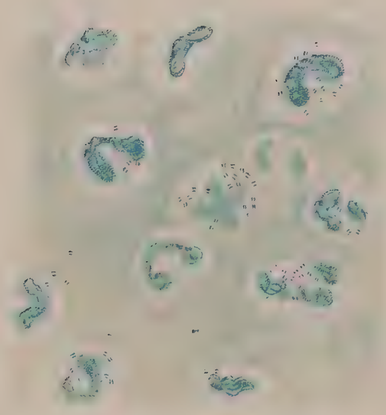
a Conjunctivitis, due to *Koch-Weeks'* bacillus.



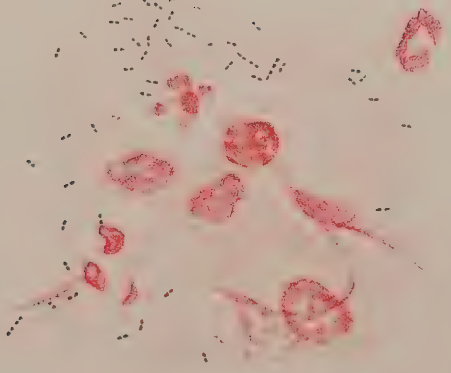
b Conjunctivitis, due to a Diplobacillus.



c Bacilli xerosis.



d Conjunctivitis gonorrhoica.



e Smear, from an ulcus serpens.

which alternates from time to time with a feeling like that produced by a foreign body, or with stinging pains and great pressure. The secretion of the eyes gradually increases until the ordinary picture of a catarrh is presented. These troubles persist into the summer, are less marked during cool and wet days and weeks, and usually pass away as summer draws to an end, the cooler months come and the sun loses its power.

In both eyes peculiar prominences of the bulbar conjunctiva can be seen in the region of the palpebral fissure, sometimes extending about its periphery, that have a yellowish gray gelatinous appearance and can be recognized to be individual formations. At the margin of the cornea these proliferations present a steep declivity, although they slope away gradually on the other side until they blend with the conjunctiva bulbi. Their surfaces are dull in places, which resemble spots of xerosis; but toward the cornea they become more transparent, and show no signs of dryness. Single broad and tortuous vessels may be seen about these elevations.

Another marked symptom is a peculiar appearance of the eye as a whole, which can be readily recognized after it has once been seen. The upper lid droops so as to give the eye a dull, cloudy appearance, while the conjunctiva assumes a peculiar discoloration; the color is changed from bright red to a smoky pale red inclining to violet, it may be said to look as if it had had milk poured over it. This discoloration is particularly marked in the palpebral conjunctiva, which exhibits characteristic lesions, while the transition folds remain normal.

The conjunctiva of the lower lid appears to be simply thickened, but that of the upper lid presents roundish, flattened prominences which may be very numerous and packed closely together, so as to make the surface look as if covered with paving stones, or may be found singly. These elevations cease at the margin of the tarsus, and are altogether absent in the transition fold, a point of importance in the differential diagnosis.

The secretion of the conjunctiva, which is free only at times, is also very peculiar. It may be found only in the transition folds in the form of long threads that can be removed with forceps. Sometimes it can be detected only after prolonged irrigation when many such threads come into view.

This is the characteristic picture of vernal catarrh, a name that is suitable only in so far as it is indicative of the recurrence of the symptoms at a certain season of the year, for even in the cold months, when the symptoms are in abeyance, the alterations in the conjunctiva remain unchanged.

The association of the changes at the limbus with changes in the palpebral conjunctiva is met with in only one group of these cases, the *mixed form of vernal catarrh.* The proliferations may occur in the marginal portion of the limbus alone while the palpebral conjunctiva remains uninvolved, the *bulbar form.* In still other cases the palpebral conjunctiva may be the only part involved and exhibit the characteristic milky cloudiness and paving stone appearance; this form is termed the *palpebral.* The mixed form is the more common. The disease is rare on the whole, though met with rather frequently in certain localities.

Vernal catarrh attacks males more commonly than females, usually appears during youth, produces its symptoms with great regularity for many years and then gradually passes away. Cases have been known to last for twenty years. The disease may also appear during middle life. The alterations in the conjunctiva are the same in winter as in summer, the symptoms alone are far less pronounced. As the disease dies out, after the lapse of a varying number of years, these alterations at the limbus and in the tarsal conjunctiva gradually undergo involution until only a slight thickening is left at the limbus, but in many cases a peculiar corneal opacity that closely resembles the arcus senilis remains. It runs parallel to the margin of the cornea in the form of a narrow band, leaving, just as in arcus senilis, a narrow, transparent zone between it and the limbus. This opacity may form a complete ring, when the proliferations formerly surrounded the entire cornea, or it may form only a sector of a circle. A certain discoloration and thickening of the mucous membrane persists after the proliferations on the tarsal conjunctiva have undergone involution and for a long time the eyes continue to have a peculiar, sleepy appearance. These symptoms, when taken together, suffice to show that a patient has suffered from vernal catarrh.

The prognosis is good, except for the long duration of the disease. Corneal complications occur only in the rarest cases. Occasionally the proliferations at the limbus involve the cornea and result in a pale, gelatinous layer on its surface.

Deviations from the normal course are also met with occasionally. Papillary elevations have been observed instead of the ordinary proliferations at the limbus. The regular intermission during winter may be omitted, or the disease may be at its height during the cold months. Sometimes a peculiar brown discoloration has been observed on the conjunctiva bulbi. Even when the disease is completely extinct, a peculiar, vascular, glassy layer can be detected on the conjunctiva by means of *Zeiss*' loupe, and very minute thickenings persist also in the cornea at the limbus.

Under the microscope the proliferations in the conjunctiva tarsi are seen to consist of the proliferated stroma of the conjunctiva. The milky discoloration of the mucous membrane is due to a thickening of the epithelium, and especially to a peculiar sclerosis of the proliferated subepithelial connective tissue. There are also vascular changes in the small arterioles at the bases of the papillæ. The proliferation of the connective tissue seems to be primary and more important than that of the epithelium in vernal catarrh. All the elements of the conjunctival tissue, the supporting tissue and the vessels, proliferate. The lymph cells generate no lymph follicles, but many plasma cells. The proliferations at the limbus are likewise of connective tissue.

No definite cause can be assigned to this disease. No specific agent has yet been detected, and still we feel compelled to think that a change takes place throughout the entire organism. These patients have a peculiar pale appearance, although no marked deviation from the normal has yet been found in the amount of hæmoglobin in the blood. It may be that further investigation will reveal new conditions. Recently it has been claimed that vernal catarrh is a "light disease," and may be caused by the action of the ultraviolet rays. *Kreibich* has pointed out the relationship of vernal catarrh to the sunlight dermatoses, summer prurigo and erythema solare, but these dermatoses are met with only exceptionally in the spring. Vernal catarrh is not cured by the withdrawal of ultraviolet light, so whether it is a "light disease" or not is as yet uncertain. Under certain unknown conditions of predisposition light may be said to be an active factor in its production. It has certainly been observed that the symptoms abate somewhat when light is excluded from the eyes, so glasses may be ordered that will absorb to a great extent the ultraviolet rays. It is not certain that a therapeutic effect is obtained in this way because both mild and obstinate cases are met with, and patients recover readily in the mountains and in winter, where the chemically acting rays of light are still active.

The palpebral form of this disease must be differentiated from trachoma, which does not attack as young people, as a rule. In vernal catarrh the changes in the conjunctiva do not invade the transition folds, while in trachoma these folds are the site of the principal changes. The "paving stones" in vernal catarrh differ altogether in appearance, form and consistence from the granules and papillæ of trachoma. The peculiar livid discoloration of the conjunctiva characteristic of vernal catarrh is never seen in trachoma, so when this is present and associated with proliferations at the limbus trachoma is at once excluded. *Herbert* first called attention to the fact that the secretion in vernal catarrh as a rule contains very many eosinophile cells, while such cells are by no means so frequent in that of trachoma.

The bulbar form of vernal catarrh may need to be differentiated from eczematous efflorescences, or phlyctenules, at the limbus. Phlyctenules develop rapidly and eyes thus affected usually present other corneal diseases associated with scrofulosis; they ulcerate quickly and become resorbed, while in vernal catarrh the proliferation at the limbus forms a firm mass which never breaks down. In rare cases a tuberculoma at the limbus may present the picture of an atypical vernal catarrh, when the microscopic examination of an excised portion of the tissue may be necessary to clear up the diagnosis.

Although the prognosis of this disease is good our therapy is impotent to control it; we can only try to alleviate the symptoms. On the theory that the ultraviolet rays form an important factor in the production of the photophobia protective spectacles may be worn, made from such glasses as *Hallauer's*, or the euphos glass recommended by *Schanz* and *Stockhausen.* When anæmia is present suitable treatment with iron and arsenic is to be strongly recommended.

When the secretion happens to be considerable the conjunctival sac should be irrigated regularly with a solution of boric acid, or a weak solution of potassium permanganate. The patients, or their relatives, learn very easily how to perform these irrigations. The removal of the secretion causes the immediate disappearance of the feeling of pressure and of the presence of a foreign body. Occasionally an application of the solution of silver nitrate may be necessary, but as a rule all irritant applications are to be avoided.

Solutions of cocain to which adrenalin has been added, weak dilutions of acetic acid (1 drop of dilute acetic acid in 10 or 20 grams of water), and a 1% solution of ichthyol, have been recommended to relieve the constant burning and itching. Solutions of bichloride of mercury, 1 : 1000 to 5000, yellow mercuric oxide ointment, ichthyol ointment, white precipitate ointment and xeroform are among the remedies that may be tried by turn during the long course of this disease, but it is doubtful if any of them produce any notable result. In many cases it becomes necessary to excise the great proliferations at the limbus, but this does not prevent recurrence. When the proliferations on the upper lid are excessive, they may be excised together with a strip of the tarsus.

17. Pterygium and Pinguecula

Sometimes a triangular membrane is to be seen on the surface of the eye exposed by the opening of the palpebral fissure, extending horizontally from the conjunctiva bulbi over the cornea, to which it is firmly attached. Its apex is rounded and directed toward the centre of the cornea, its base is lost in the conjunctiva bulbi near the equator of the eyeball, and in it run numerous bright red vessels that are distinctly visible. The veining of this membrane gives it a certain resemblance to the wing of an insect, whence it has derived its name of fly wing, or pterygium.

A pterygium is a fold of conjunctiva that has grown over upon the cornea. This can be shown in the cocainized eye by passing a probe beneath it near the limbus, from both above and below, into a blind cul de sac. It is also evident from some fine grooves on its otherwise smooth surface. The upper and lower cul de sacs end at the margin of the cornea where the pterygium is firmly adherent. Toward the equator the pterygium gradually blends with the surrounding tissue; frequently it extends into and involves the plica semilunaris.

The portion of the pterygium that lies on the cornea is called its head, that lying in the conjunctiva its body and the intermediate portion its neck. It develops slowly until its progress comes to a standstill, and it is important to be able to determine in any given case whether the condition is progressive or stationary.

As long as a pterygium has a tendency to grow the head presents a gray border, which is without vessels and looks peculiarly voluminous and gelatinous, but when the process has been arrested this border becomes thinner and assumes an aponeurotic appearance. As long as advancement continues the entire body of the pterygium is large and hyperæmic, but when this has ceased, the vessels retrogress, the body becomes thinner and appears more tendinous. The usual site of a pterygium is on the nasal side of the cornea, they are met with less often on the temporal side, and sometimes two pterygia appear at the same time in one eye. In rare cases four pterygia have been observed in one and the same person. It occurs in men twice as often as in women and is usually met with in persons over forty years of age.

It may be mentioned that in rare cases cysts of various sizes have been met with in the head, or neck, of a pterygium. These are retention cysts, are filled with a clear fluid, and originate from little glands and epithelial depressions. Perhaps they may also originate from dilated lymph vessels.

The troubles induced by a pterygium are varied. As soon as the apex reaches the pupillary region vision is interfered with to a degree which increases as the centre of the cornea is approached. By its presence it irritates the eye and causes catarrhal trouble. When it is so large as to involve the plica semilunaris the lacus lacrimalis is interfered with and epiphora is produced. Motor disturbances and diplopia may result when the conjunctiva is so tense that the opposite muscle is unable to overcome its resistance. Add to these troubles the cosmetic effect produced by a progressive, vascular pterygium, and the desire of the patient to have it removed can be readily appreciated.

In order to understand the development of a pterygium it is necessary to study another condition in which a peculiar change takes place in the conjunctiva bulbi close to the cornea in the palpebral fissure.

A round, oval, or triangular elevation, of a grayish yellow color and with a somewhat dull surface, is formed at this place. About it run some rather hyperæmic conjunctival vessels. The conjunctiva is thickened and not rarely extends over the margin of the cornea. This is called a pinguecula. It is of interest for various reasons, chiefly because from it the pterygium develops.

A pinguecula appears at first on macroscopical examination to be a spot, but with a loupe it can be seen to be composed of a number of spots. Like pterygium it is met with more frequently on the nasal side of the cornea than on the temporal, but it may be found on both sides and in both eyes. It is always situated in the horizontal diameter of the zone of the palpebral fissure near the margin of the cornea. It is met with very frequently, especially in people over forty years of age. Occasionally a subconjunctival hemorrhage from other causes near the margin of the cornea will bring into view, by contrast, the primary stage of a pinguecula that had not been seen before. It is met with more often in men than in women and does not appear to be capable of involution.

The name pinguecula is a misnomer. The microscopical examination of its tissue shows that it is caused not by a fatty, but by another form of degeneration. The conjunctiva is thickened, its epithelium appears to be essentially normal, though in rare instances proliferative processes have been found in it, while the connective and subconjunctival tissues are, on the contrary, much changed. The connective tissue fibres are thickened and have undergone hyalin degeneration, in which the elastic fibres of the submucosa are also involved, according to *Huebner*. Free hyalin is also present. The thickened connective tissue fibres curl together in a peculiar manner. Its yellow color is due partly to the degenerated elastic fibres, partly to the free hyalin.

The peculiar loçalization of this degeneration of the conjunctiva indicates its origin. The zone of the palpebral fissure is the most uncovered and unprotected portion of the conjunctiva, where external causes of irritation, such as atmospheric influences, can act most energetically; hence pinguecula is met with most frequently among people who are much exposed to such influences, like coachmen and farmers. Evidently there is an individual susceptibility to this condition, as otherwise it would be seen with much greater frequency. It is not uncommon to see it in one eye alone. Senile degeneration cannot be said to predispose to this condition and apparently the causes of irritation must act for a long time to produce this hyalin degeneration, as it occurs mainly in persons over forty years of age, but pinguecula is sometimes met with in young people, and its essential nature is still unsettled. But one thing is certain; this degeneration of the conjunctiva does not always remain stationary, it has a causal connection with pterygium.

Fuchs says that pterygium is a proliferation of pinguecula over the cornea. It is certainly not a simple proliferation and encroachment of the conjunctiva, for a pinguecula gradually disappears when it is replaced by a pterygium. The anatomical examination reveals bits of tissue that originate in a pinguecula only in certain early stages of pterygium, in all the later stages it is composed of the tissues of the conjunctiva bulbi; the epithelial coat, the goblet cells and the stroma are the same as those of the conjunctiva. Wherever the pterygium becomes adherent to the cornea *Bowman's* membrane is destroyed; the anterior margin of the pterygium itself penetrates between the superficial layers of the cornea. How the transition of the pinguecula to the cornea takes place is still unknown, but corneal ulceration at the head of a pterygium, such as was formerly believed to exist, is never found. The etiologic connection between pinguecula and the pterygium that develops from it, which is called a true pterygium, explains why the latter always grows in the zone of the palpebral fissure. It occupies almost invariably a horizontal position, and even when its position is oblique it always lies in this zone with its head beneath the horizontal meridian of the cornea.

The Pseudopterygium

The adhesion of a fold of conjunctiva to the cornea may result from injuries, burns and ulcerations. When an area on the surface of the cornea has been eroded, as by a lime burn, or an ulcer, it may happen that a fold of chemotic conjunctiva comes to lie upon it and the inflammation present will then cause the two surfaces to adhere. The condition thus produced is known as a false, pseudo-, or cicatricial pterygium. In practice they are met with principally after lime burns, but sometimes a chemotic fold of conjunctiva becomes attached in this way to an ulcer of the cornea in the course of a blennorrhœa, or some other inflammation.

As the defects produced by burns and ulcers are not confined to any particular part of the surface of the cornea it follows that pseudopterygia occur in all parts of that surface, while true pterygia are met with only in the area of the palpebral fissure. Naturally the form of the pseudopterygium is not as regular as that of the true; it is also apt to be surrounded by extensive leucomata of the cornea, while with a true pterygium, although the gray margin of the head is present, the rest of the cornea remains perfectly clear.

In burns a great part of the conjunctiva is often destroyed, or at least very badly injured, so an adhesion between the anterior and posterior walls of the conjunctival sac, a symblepharon, is frequently formed simultaneously with a pseudopterygium. The pseudopterygium is not progressive, because its extent is defined from the first by the destroyed portion of the corneal surface. Finally in many cases a probe can be passed beneath a cicatricial pterygium and the fold be lifted up, a feat that cannot be performed upon a true pterygium.

Operative removal of a pterygium. After the eye has been cocainized the neck of the pterygium is seized and lifted up with fixation forceps, then the head is carefully dissected off the cornea with a keratome, or *Graefe* knife. The most important part of the operation is to remove as thoroughly as possible all abnormal tissue, especially the gelatinous margin. Two convergent incisions are then made into the body of the pterygium from its upper and lower margins, thus removing a rhomboidal piece of the growth. The gaping wound in the conjunctiva bulbi is closed with two sutures after the conjunctiva on each side has been freed somewhat from the episcleral tissue with scissors. The surface of the wound on the cornea is left alone, unless the removal of the growth with the knife has been incomplete, or a recurrence is feared, when it may be cauterized. This surface heals quickly leaving a cicatrix. Even when this operation, devised by *Arlt*, is performed with great care it is always possible for a recurrence to take place after a time. An ointment should be applied to the eye for a few days and the sutures should be removed at the end of three or four days.

18. A Conjunctivitis from Caterpillar Hairs

A gentleman is walking in a wood when he is struck in the eye by a caterpillar; a lively conjunctivitis soon develops which presents unusual and typical features. It is a well-known fact that caterpillar hairs can produce wheals and burning exanthemata in the skin, but the reaction produced by them in the eye is extremely severe. The lids become considerably swollen, the conjunctiva becomes reddened and yields an abundant mucopurulent secretion. Thus far the picture is simply that of an acute conjunctivitis; the characteristic peculiarity is the presence of some grayish red, or yellow nodules, about as large as millet seeds, in the conjunctiva bulbi, usually in its lower part. From these nodules *Sæmisch* named this form of conjunctivitis ophthalmia nodosa. Each nodule contains a fine caterpillar hair, which may sometimes be seen with the aid of *Zeiss'* loupe. When the patients come under observation sufficiently early the prognosis is good.

The conjunctival sac is to be thoroughly cleansed in order that no other caterpillar hairs may be left; if a hair can be seen to be protruding from a nodule it may be withdrawn, otherwise each nodule is to be excised.

Anatomical examination shows that these nodes resemble tubercular nodules; beneath the thickened epithelium is found an inflammatory infiltration of the connective tissue, particularly about the vessels, in which the lymphoid and giant cells are heaped up, but instead of tubercle bacilli each contains a caterpillar hair.

The nodes are surrounded by a cellular layer of epitheloid character. The clinical picture of ophthalmia nodosa is differentiated from that of tuberculosis of the conjunctiva by the facts that the former appears by preference in the conjunctiva bulbi, while the latter are usually situated in the conjunctiva tarsi, and that the inflammation engendered by caterpillar hairs never leads to ulceration and necrosis.

The fine caterpillar hairs penetrate the mucous membrane very easily. The procession caterpillar, a species of the Bombyces, is the most common cause of this disease in Europe, but other species that have prickly hairs have been known to produce it. *Schmidt-Rimpler* and *Markus* have described cases in which the inflammation had been produced by the hairs of plants. When the inflammation is neglected, or is not recognized in time, the prognosis is not so good. The hairs may pass into the cornea and sclera, or even through these membranes into the iris and chorioid, where they set up very grave diseases, likewise associated with the formation of nodules. In the conjunctiva itself the nodules may spontaneously undergo involution and disappear; some observers have assumed that the hairs had been extruded in such cases, others, for example *Stargardt*, have seen a dissolution and resorption of the same under the microscope.

19. Reflections Concerning Differential Diagnosis Suggested by Chemosis and Ecchymosis of the Conjunctiva

Exudations of serum and of blood beneath the conjunctiva are met with frequently. The exudation of serum beneath the conjunctiva bulbi and the transition folds is called chemosis; the palpebral conjunctiva is not elevated in like manner because it is firmly attached to the tarsus. The exudation of blood beneath the conjunctiva is termed ecchymosis, or hemorrhage. As both conditions are met with in a great variety of diseases and are of some diagnostic value it may be well to study them with reference to the groups of cases in which they occur.

I. Description of Chemosis

In a case of commencing panophthalmitis the conjunctiva bulbi is raised up until it partly overhangs the cornea. An œdematous fold of conjunctiva may be pinched between the lids. The serous transudate shines through the conjunctiva and is sometimes of a yellowish color, at other times not so yellow, but clear and watery.

When the conjunctiva bulbi is seen to be thus lifted from its bed by serous or aqueous fluid, the first thought is naturally whether such an accumulation of fluid has not been produced by an escape of the aqueous into the subconjunctival space through an opening into the anterior chamber, but such an opening, covered only by the conjunctiva, is met with only after accidents or operations. Sometimes a little transparent elevation of the conjunctiva near the cicatrix can be seen after an operation for the removal of cataract, a condition that is known as a cystoid cicatrix. Such a condition is brought about when a bit of iris is caught between the lips of the wound and prevents a firm union.

This peculiar form of œdema, or chemosis, has been given the name of filtration œdema of the conjunctiva. When it has once been seen the cystoid cicatrix will not be mistaken for any of the other forms of chemosis; it forms a local elevation of the conjunctiva confined to a small area, while in all other forms of chemosis the elevation of the membrane is more diffuse. If, in any given case, this form can be immediately excluded by the history and a single glance, there will remain only three principal causes of chemosis to be considered.

Group I. Chemosis in Acute Inflammations

The exudation of serum beneath the conjunctiva occurs 1, in acute inflammatory hyperæmias; 2, in cases of obstruction to the outflow of blood and lymph from the conjunctiva; 3, in certain changes in the composition of the blood. Chemosis of the conjunctiva is one of the most frequent symptoms of acute inflammation of the eye and when in any particular case the cause has been determined to be an acute inflammation its seat remains to be found.

1. The inflammation may be seated in the conjunctiva itself, in which case even a trifling inflammation may cause a slight œdema. This œdema is one of the cardinal symptoms of an acute conjunctivitis. It is met with in severe cases induced by the pneumococcus and the *Koch-Weeks* bacillus and still more markedly in conjunctivitis gonorrhoica and crouposa. Hence the first thing to determine is whether the chemosis is due to some form of acute conjunctivitis.

2. If the cause is not to be found in the conjunctiva it is to be sought for next in the interior of the eye, for acute purulent inflammations, particularly of the uveal tract and vitreous, as well as the panophthalmitis from infected perforating wounds, are associated with great chemosis. In these cases the substances that excite inflammation press along the vessels in *Tenon's* space and produce the œdema.

Frequently a severe purulent inflammation of the cornea excites chemosis, as in grave cases of ulcus serpens.

Very similar conditions are produced when the pyogenic agents enter the eye not through a perforating wound, but by way of the blood, as in the metastatic ophthalmia of sepsis and puerperal pyæmia.

An intraocular process not of bacterial origin can also produce chemosis, as in acute inflammatory glaucoma, in which it is caused by the inflammation and stasis. It behooves one to think quickly in the presence of a chemosis.

3. Inflammation of the tissues about the eye may also excite chemosis. A simple hordeolum near the outer canthus may extend its inflammatory action so far that the entire temporal portion of the conjunctiva may be chemotic. A phlegmon of the lacrymal sac may produce a similar condition, and in facial erysipelas not only the lids, but the conjunctiva as well, are œdematous. A periostitis of the margin of the orbit, and still more an orbital phlegmon, may be the cause of a high degree of chemosis, and occasionally its origin is to be found in the cranial cavity. The presence of a chemosis of the conjunctiva in cerebrospinal meningitis was first observed by *v. Leyden;* it happens rarely, but it may be of service to the physician to take into account both the pupils and the conjunctiva when he tries to make an early diagnosis of meningitis, a diagnosis that is not always to be made easily. In such cases we must suppose that the inflammatory products pass through the orbit and are deposited beneath the conjunctiva.

Group II. Chemosis from Stasis of Blood and Lymph

As compared with the numerous cases in which chemosis is caused by an acute inflammatory process those in which it results purely from stasis are few, but the possibility of their occurrence must be taken into account.

Sometimes when a large retrobulbar tumor presses heavily upon the eyeball a chemosis is caused by the interference with the outflow of blood from the orbit.

Occasionally a chemosis is met with in old people who have been exposed for some time to the weather, as after a storm; this may be considered to be a disturbance of the lymph circulation. I have seen an old sailor, who had otherwise perfectly healthy eyes, develop such a chemosis of the lower transition fold during storms. *Spicer* described a chemosis that lasted for months and was ascribed to an obstruction in the efferent lymph channels.

To this group, which is for the most part caused by lymph stasis, is to be added the chemosis brought about in the eye for therapeutical purposes in attempts to clear up opacities of the cornea. The transudate that is produced by the inflammatory effect of the irritant becomes stasic and remains in the eye for a longer or shorter period. If a 2 to 5% solution of dionin is instilled into the eye of a patient with an opacity of the cornea, a tense chemosis of the conjunctiva is formed in a few minutes, preceded by a feeling of burning and an increasing injection of the eye. This œdema passes away again in a few hours and we hope to obtain a beneficial effect on the corneal opacity through the influence produced on the lymph circulation.

Group III. Chemosis From Changes in the Composition of the Blood

Sometimes chemosis is met with in persons suffering from anæmia or chlorosis, when other and more important symptoms call for an examination of the blood. A transient, recurrent chemosis may lead to the examination of the urine, for this symptom happens, though rarely, in *Bright's* disease. Such a chemosis, that comes and goes, is called œdema fugax. A regularly recurring chemosis in women may be connected with menstruation, and chemosis may also be produced by urticaria. *De Schweinitz* met with a chemosis in the course of a trigeminal neuralgia, and I have myself observed a regularly recurring chemosis in one such case associated with marked attacks of migraine.

The observation of this simple phenomenon of the eye leads the physician diagnostically through the most varied regions; it is the same with subconjunctival hemorrhage.

II. Description of Subconjunctival Hemorrhage

Hemorrhages into or beneath the conjunctiva are extremely common. The general appearance may be drawn from the description of our first case. In the nasal half of the conjunctiva bulbi is a place which presents all shades from bright red to dark and is sharply defined at its periphery. To differentiate it from an injection of the conjunctiva it may be noted that injected vessels are not visible in the lake of blood and that a hemorrhage is more diffuse.

In such a case a pinguecula may define itself distinctly as a bright, or whitish yellow spot on the red background. The conjunctiva is firmly adherent to the limbus at the margin of the cornea, so that it cannot be raised by the blood, and therefore the hemorrhage at this place has a narrow gray edge. Yet in such cases some blood, and more hæmoglobin, penetrates into the cornea by diffusion, where it can be seen in a thin layer between the lamellæ. The contiguous margins of the cornea may receive a greenish discoloration and this layer of blood may cause the iris in blue eyes to appear green.

As serum can accumulate beneath the conjunctiva in acute conjunctivitis so the vessels become permeable to the blood cells in inflammatory hyperæmia. Chemosis and ecchymosis are therefore frequently associated, and, as in chemosis, acute conjunctivitis is to be thought of first when an ecchymosis of the conjunctiva is met with. Not only in pneumococcal, but in other forms of acute conjunctivitis, numerous little spots of blood are to be seen, particularly in the upper half of the conjunctiva bulbi. In a croupous conjunctivitis the conjunctiva bleeds very easily when the membrane is removed. These hemorrhages in acute inflammation of the conjunctiva are usually in the form of little spots and are easily recognized.

Injuries may be said to be the second most important and frequent cause of conjunctival hemorrhages. Subconjunctival hemorrhages may be produced by three classes of traumatisms to the eye itself, contusions, perforations and superficial injuries. These include operations performed on the eye; the operation for strabismus may cause very large suffusions.

Subconjunctival hemorrhages may be produced also by injuries which do not affect the eye itself. Very extensive ones are often met with after fractures of the base of the skull and fissures of the roof of the orbit and of the sphenoid, when the latter extend to the bony walls of the orbit. Clinical experience has shown that in fissures of the sphenoid subconjunctival hemorrhages are apt to appear later in the temporal portion of the conjunctiva bulbi, and it happens not infrequently that the probability of the diagnosis is increased by waiting a while until the blood has appeared beneath the conjunctiva.

The surgeon sometimes sees effusions beneath the conjunctiva in connection with grave injuries of the thoracic and abdominal cavities, as for example in railroad accidents. In these cases other ecchymoses are usually present in the skin of the head and face, which are caused by the sudden production of a stasis in the jugular veins as the result of the compression. But such violent compressions of the body are not necessary in order to produce congestions of the blood in the head, and stases in the jugular veins. Vomiting, sneezing, blowing the nose, lifting of heavy weights, active labor pains, all of these may cause subconjunctival hemorrhage. Cough is so common a cause that the first question put to a patient with a subconjunctival hemorrhage is apt to be whether he has a cough. In whooping cough these ecchymoses are very common, they may appear in both eyes and be quite extensive. In the overwhelming majority of cases these subconjunctival hemorrhages pass through a series of color changes and disappear. No special treatment is necessary, though it is well to order some eye lotion in order to comfort the patient.

In rare cases of whooping cough the hemorrhages take place into the orbit and produce marked exophthalmos. Once I succeeded in a fresh case of this nature in evacuating the still fluid blood from the orbit by puncture. Hemorrhages into the anterior chamber and into the retina have been described not infrequently, so it is well for the physician to examine ophthalmoscopically children with pertussis. Other serious disturbances of vision that may develop in the course of a whooping cough are homonymous hemianopsia and paralyses of various nerves from hemorrhages in the region of their nuclei.

Finally subconjunctival hemorrhages may depend on derangements of the entire organism. The brittleness of the vessels in old age, arteriosclerosis, can produce such hemorrhages either with or without any other cause. A man who was much frightened because of such an ecchymosis consulted me; I reassured him, although he had arteriosclerosis, but he had hardly reached home when he had an attack of apoplexy that proved fatal. Even though such an experience is exceptional, it serves to impress the fact that the conjunctival hemorrhage is an eye symptom indicative of the general condition.

Conjunctival hemorrhages occur in all general diseases that predispose to hemorrhage, such as diabetes, scurvy, purpura hemorrhagica, malaria, etc.

At the suggestion of chemosis and subconjunctival hemorrhage we have thus made a little excursion into differential diagnosis which may later prove of advantage. Attention to the minutest detail makes the good diagnostician.

By way of supplement it may be mentioned that air sometimes penetrates beneath the conjunctiva. This emphysema of the conjunctiva is usually associated with emphysema of the lids. When a communication between the orbit and the nasal cavity is established by some injury, air can be forced on expiration into the spaces of the orbital and subconjunctival tissues. When the air is driven into the orbit exophthalmos can readily be produced. The condition is easily recognized by the feeling of crepitation communicated to the fingers when pressure is made on the lid. This emphysema is prone to follow injuries at the inner canthus that involve the thin lamina papyracea. If left quietly alone the air becomes resorbed very soon.

20. The Benign Tumors of the Conjunctiva

Tumors of the eye may be both intra- and extraocular. We regard intraocular growths with anxious eyes, because they are usually found in practice to be malignant, in which case the only help is in the early and prompt removal of the entire eye. But many of the extraocular tumors are not malignant and the most important question that arises clinically, when an extraocular tumor of the eye comes under observation, is whether it is malignant, or benign. Next comes the question whether it is congenital or not. Two things are necessary in order to obtain an answer to the first question; an accurate clinical examination of the particular case, and a knowledge of what tumors of the conjunctiva are benign and what malignant.

Case of Dermoid of the Conjunctiva

At the lower, outer margin of the cornea of this patient is a hemispherical prominence as large as a small bean, of a whitish red and also whitish yellow color, situated partly in the conjunctiva, partly in the cornea. Its surface appears rather dry, resembling epidermis, and it is provided with minute hairs which are more or less fine. The suggestion comes almost spontaneously that this is a dermoid of the conjunctiva, and when the history shows that the tumor is congenital the diagnosis is positive. Dermoids are congenital without exception. Clinical experience has proved them to be benign; when the eye has attained its full growth the dermoid has also reached its greatest extent and remains stationary. In only very rare cases do these growths increase in size and proliferate over a large portion of the cornea.

I. The Different Kinds of Dermoids

There are three distinct kinds of dermoids: 1, those met with at the corneoscleral margin; 2, dermoids of the transition folds; 3, dermoids of the caruncle.

1. The Dermoid of the Corneoscleral Margin

This may be met with in any part of the corneoscleral margin, but it exhibits a certain preference for the lower, outer portion. It is usually single, but may be double and may be present in both eyes. Such growths are not infrequently seen in the eyes of such animals as calves, cattle and dogs.

2. The Dermoid of the Transition Folds

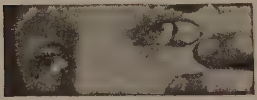


Fig. 16.—Lipodermoid of the Conjunctiva.

This form of dermoid is found in the subconjunctival tissue toward the transition folds. Its favorite seat is upon the surface of the eyeball between the external and superior recti. Sometimes it develops in the palpebral fissure itself and can be seen when the eye is turned inward. This dermoid contains more fat than the one just described, so that it is softer. It also frequently increases in size by the addition of fat. It is freely movable over the subjacent tissues, but the conjunctiva is attached to its surface. Because of the great amount of fat it contains it has also been designated lipodermoid.

3. Dermoid of the Caruncle

In very rare cases dermoids occur in the caruncle and plica semilunaris and extend from there to the cornea.

II. Occurrence of Other Developmental Anomalies

As the dermoid is a congenital tumor it is not surprising that it is sometimes associated with other anomalies of development either in the eye or elsewhere. Among such anomalies may be mentioned lipomata, warts, ichthyosis and appendices auriculares, as well as colobomata of the lids, iris and chorioid, microphthalmos, ectopia of the pupil, etc. The presence elsewhere of such developmental anomalies aids in the diagnosis of the dermoid nature of the growth.

III. Anatomy and Origin of the Dermoid

What impresses one most about these tumors is their close relation to the skin. In a dermoid of the corneoscleral margin the epithelium over the growth is thick and horny; beneath this lies a dense, fibrous connective tissue containing hair bulbs and sebaceous glands. The transition into the cornea and sclera is quite gradual. Sweat glands, smooth, transversely striated muscles and even cartilage have been found in them. In the subconjunctival lipodermoid the condition is analogous, except for the excessive amount of fat. Theories are much divided concerning the origin of dermoids. Whether it is due to a fœtal invagination of the external blastoderm, as *Remak* believes, or whether *v. Duyse* is right in believing that the cause is an adhesion of the amnion to the surface of the eyeball in a very early period of fœtal life, we do not know. Embryonic skin tissue must at any rate remain on the surface of the eyeball.

IV. Therapy of Dermoid

Although a dermoid is a benign tumor it should be removed, not only to improve the cosmetic appearance of the patient, but because the hair on its surface forms a source of irritation to the eye. The dermoid is dissected out from the cornea and then the attempt is made to shell it out of the conjunctiva. The defect left is covered by drawing together the edges of the conjunctival wound.

It has already been said that the differentiation between a benign and a malignant extraocular tumor of the eye depends not only on the accurate clinical examination of the particular case, but also on a knowledge of what benign and malignant tumors may be met with. The benign tumors that occur in the conjunctiva, in addition to the dermoid which has just been mentioned, are angiomata, osteomata, polypoid growths, cysts and nævi.

Angiomata of the Conjunctiva

An angioma is composed of vessels that are either newly formed, or have undergone extensive changes in their walls, and, when met with in the conjunctiva, may be recognized immediately by its dark red, or blue red color. It may be in the form of a teleangiectiasis, or of a cavernoma, just the same as elsewhere in the body, and it may occur primarily in the conjunctiva, or it may involve that tissue by an extension from the skin of the lid. It is a growth that is rarely seen, is usually situated near the inner canthus and is movable with the conjunctiva. Its commencement is ordinarily congenital and it is usually noticed by the parents at an early period. Many procedures have been recommended for its removal, the most suitable is to pass a ligature about it in the deep tissues and to follow this by excision. Lymphangiomata very rarely have been seen in the conjunctiva; more frequently they extend from the lids and orbit, and encroach on the conjunctiva.

Osteomata

Osteomata of the conjunctiva are extremely rare. Those which have been met with were smooth and situated between the external and superior recti, like the lipodermoids. They were congenital and the eyes in all other respects were perfectly well. Histologically they consisted of connective tissue capsules, each containing a scale of true bone. They can easily be removed and do not recur.

Polypoid Tumors

Polypoid growths are met with more frequently than any of the tumors that have yet been described. In general they are outgrowths that resemble polyps and are attached to the conjunctiva by means of pedicles. They occur on the conjunctiva tarsi, in the transition folds and in the caruncle, and may be divided into three groups.

First are those that are to be classed as fibromas. These are met with usually on the palpebral conjunctiva, may grow rapidly, and have a great tendency to spontaneous hemorrhage, which may be very severe, as *Elschnig* observed. They should be removed and their bases should be cauterized. Other fibromas of a harder consistence are sometimes found in the caruncle, where they may attain a considerable size.

The surface of a papilloma of the conjunctiva is not smooth, like that of a fibroma, but is fissured so as to make the growth resemble a raspberry, or cauliflower. This characteristic configuration is the result of a papillary proliferation carried on step by step. They are covered with a thick layer of epithelium. They are met with chiefly in males and have been known to undergo carcinomatous degeneration. The possibility that this last may happen is alone sufficient to justify their radical extirpation.

The third and last group of polypoid growths of the conjunctiva is composed of the so called granulation tumors. In contrast with other polypoid tumors these are not covered by conjunctiva, but consist of granulation tissue. They develop in places where the conjunctiva has suffered a loss of substance and are therefore met with occasionally after operations for strabismus, when the wound in the conjunctiva has not been carefully sutured, and after enucleations in the bottom of the conjunctival sac. When a chalazion ruptures through the conjunctiva masses of such granulations proliferate from the opening. The removal of these growths presents no difficulties.

Cysts of the Conjunctiva

A prominence in the conjunctiva, sharply delimited from the surrounding tissue, with watery, or bright yellow contents shining through the thin wall, is a cyst of the conjunctiva. A cyst may be produced in various ways and the different varieties appear by choice in certain places.

First Group of Cysts

Cysts belonging to this group arise from ectatic lymph vessels and are therefore called lymph cysts. They may be congenital, or they may develop in later life, and are usually situated in the conjunctiva bulbi. Their superficial position and movability upon the sclera are indicative of their origin. Sometimes a dilated lymph vessel can be observed to extend from the end of the cyst. Multiple ectasiæ of the lymph vessels also occur on the eyeball.

Second Group of Cysts

This group originates from pathological changes in the epithelium. When the conjunctiva is inflamed the epithelium may proliferate into it in the form of a cunicle, then the cells in the centre of such a cunicle may die, a hollow space be formed and a cyst develop. Or folds may be formed in the conjunctiva, as happens in pterygium, that come together and fuse above, thus creating hollow spaces lined with epithelium, from which cysts may proceed to grow.

Cysts may also originate from *Krause's* glands of the conjunctiva, when the openings of the glands have been closed by inflammation. These are known as retention cysts, are found in the lateral portions of the transition folds and may attain a considerable size.

Third Group of Cysts

A penetrating, or a nonpenetrating injury of the eyeball may result in the formation of a cyst. The cystoid cicatrix after operations for cataract and glaucoma is an example of the former. Such cystlike formations are particularly apt to occur when iris tissue is incarcerated in the wound, or the intraocular tension is increased. A nonpenetrating injury may likewise cause a cyst, as occasionally happens after an operation for strabismus. It cannot be determined clinically whether such cysts have been produced by the formation of folds, by the presence of a foreign body, or by epithelial cells that have been transplanted into the subconjunctival tissue. Cysticercus cysts have been observed very rarely in the conjunctiva.

Nævus of the Conjunctiva

We come now to a tumor of the conjunctiva that may readily overstep the limits of a benign character. The nævus appears clinically in two forms, pigmented and nonpigmented, each of which has clinically distinctive features.

The nonpigmented nævus is met with almost exclusively in the conjunctiva bulbi, usually close to the margin of the cornea, where it forms a flat, yellowish red, smooth prominence which can be moved about freely over the sclera.

The pigmented nævus may develop anywhere in the conjunctiva. The nonpigmented usually is met with in young people, the pigmented occurs in old people as well. In its rudimentary form it must certainly be considered congenital. Both forms may show signs of renewed activity after long intervals of quiet. The nonpigmented nævus disturbs the eye by growing over the cornea, but the pigmented nævus very readily gives rise to the malignant sarcoma. Every suspicious nævus should therefore be destroyed as early and as radically as possible. When it is situated at the margin of the cornea and is adherent, it must be dissected away very carefully.

Under the microscope it may be seen that the epithelium sends long processes into the stroma of the conjunctiva and, as a consequence, often degenerates. Between these processes cells, known as nævus cells, lie heaped together. When the nævus is pigmented the pigment cells lie in part between the nævus cells, or in the vascular intermediate tissue. Whence the nævus cells originate is still a debated question; some think they come from the epithelium, others that they originate from the connective tissue. These benign tumors form a marked contrast with the epithelioma and sarcoma of the conjunctiva, which next demand our consideration.

21. The Malignant Tumors of the Conjunctiva

An elderly man states that something has been growing over his eye for several months. It was noticed first by members of his family, but he paid no attention to it until recently, when it began to grow rapidly and to interfere with his vision.

On the inner side of the eye, near the margin of the cornea, is a flat, whitish gray, fairly firm growth which has involved a great part of the conjunctiva bulbi. There is no ulceration. The growth moves back and forth with the conjunctiva, but is firmly adherent at the limbus, whence it extends into the cornea, where it is absolutely immovable, as can be demonstrated when the eye is cocainized. It contains numerous bloodvessels.

Pannus is not so thick and presents no such involvement of the conjunctiva bulbi. The proliferation at the limbus is not that of vernal catarrh, for all the other changes and symptoms characteristic of that disease are absent. It has neither the triangular form, nor the gray, gelatinous head of the advancing pterygium; a probe cannot be introduced beneath it at the neck; it has grown more diffusely over the cornea and is firmly adherent to the subjacent tissue. Its appearance as a whole, its consistence and its irregular form and surface reveal it to be a neoplasm on the surface of the eyeball. The most important of the benign tumors of the conjunctiva are the congenital, such as the dermoid, angioma and nævus, and the acquired, including cysts and polypoid growths, but it is none of these. The thought of a polypoid tumor, or of a nævus undergoing malignant degeneration, may momentarily suggest itself, but this tumor is not pigmented, and has not the form of a polyp. If the age of the patient is taken into consideration with the facts that the tumor is growing and menacing the vision, it becomes evident that this is a malignant tumor. The only malignant tumors met with in the conjunctiva are the epithelioma and the sarcoma. In such cases the anatomical examination naturally is decisive, but the interpreta-

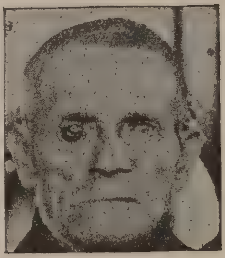


FIG. 17.
Melano Sarcoma of the Conjunctiva.

tion of the findings is often difficult. The question just now is how to establish this diagnosis clinically.

These tumors follow no fixed rules; each case must be considered individually, and yet, though they have some features in common, they possess characteristics by means of which they may be clinically differentiated when they are primary growths of the conjunctiva.

Both show a certain predilection for the limbus, because the epithelium changes its character where it passes from the conjunctiva to the cornea so that irregularities may readily happen in its development, and even normally the epithelium sends processes deep into the tissue. This predilection is in keeping with the known fact that malignant neoplasms are apt to develop in places where the epithelium of the skin passes over into that of mucous membranes, as in cases of carcinoma of the lip, nose and margin of the lid.

Several cases of malignant tumors of the conjunctiva have been described by *Hartmann*, *Manfredi*, *Schneider*, *Lagrange* and others, as the results of injuries received at the limbus, but we cannot recognize the ultimate cause of these neoplasms. We must not forget that these malignant tumors may start from other places in the conjunctiva. The predilection of the sarcoma to originate at the limbus may be partly explained also by the fact that pigment is normally present at this place in the conjunctiva; brunettes are apt to have a brown stain, sometimes dark spots, at the limbus, and, as we have already seen, true nævi occur there as well as at the lid margin.

The inclination of both of these tumors is to remain local. They recur very readily, but they rarely penetrate into the eye and still more rarely cause metastases. It is quite otherwise with intraocular sarcomas from which early metastasis is to be feared. *Grawitz* recently demonstrated a case of multiple sarcomata of the skin which originated from an unsuspected and unrecognized sarcoma of the chorioid. It is self evident that this tendency of malignant tumors of the conjunctiva to remain local for a long time must be taken cum grano salis, for eventually generalization may result from them also.

The epithelioma may contain pigment, and the sarcoma may be a leucosarcoma, and even histologically their differentiation may be difficult, as both tumors have an alveolar structure, but ordinarily the sarcoma of the conjunctiva contains pigment while the epithelioma does not. The melanosarcoma usually starts as a scarcely noticeable dark spot at the limbus and may develop from a nævus. As the amount of pigment in a nævus is apt to be not uniform, and the sarcoma is generally very vascular, its color may receive a grayish, or reddish gray tone. As the tumor spreads it remains movable in the conjunctiva. When it extends beyond the margin of the cornea it usually forms a sharp elevation and may, as *Seiderer* observed, cover the cornea for a considerable time without invading its tissue. When the cornea is invaded the sarcoma spreads beneath the epithelium leaving *Bowman's* membrane intact. It rarely, and only at a late period, penetrates into the parenchyma of the cornea and the interior of the eye. In rare cases a sarcoma may start from the cornea itself, as has been observed by *Panas*, *Gonin* and others. It likewise penetrates the sclera rarely and at a late stage; when penetration does occur the vessels at the limbus furnish the track for its passage. A former assistant of mine, *Joersz*, met with a purely subconjunctival sarcoma which probably started from the sheath of the tendon of the external rectus.

A case of this nature is almost certain to be primary, for thus far *Kawka* has described the only one in which the tumor was due to metastasis. Sarcoma of the conjunctiva is a very rare disease; 31% of all recorded cases have occurred between the ages of fifty and sixty, 15% between the ages of ten and twenty. The diagnosis of at least the pigmented sarcoma can be made clinically by taking all of these factors into account. An examination of the interior of the eye can exclude immediately the possible presence of a perforating intraocular sarcoma, or tuberculous growth from the iris. It may be mentioned that *Uhthoff* has seen a case in which particles of quartz at the limbus produced a benign, brownish yellow tumor of the conjunctiva bulbi.

The course of neglected cases of sarcoma, or of grave recurrences, may be told briefly. The growing tumor fills the conjunctival sac and causes serious, recurrent hemorrhages. When it involves the cornea vision is compromised. Finally the tumor ulcerates and protrudes through the palpebral fissure.

An epithelioma is usually nonpigmented, is flatter than an epibulbar sarcoma and lacks its abrupt demarcation from the cornea. Its surface is less smooth, but more granulated and rough, and its color is more inclined to white or grayish white. It causes less hemorrhage, even when its surface is ulcerated. It occasions swelling of the neighboring glands with comparative infrequency, and is more apt than sarcoma to cause inflammatory irritation of the conjunctiva and to interfere with the vision. As it continues to grow it involves the neighboring skin of the lid and, in advanced cases, it not uncommonly happens that all the tissues of the orbit about the eyeball become involved, and the tumor is then known as a peribulbar epithelioma.

Its behavior toward the cornea and sclera varies. Sometimes an epithelioma will remain attached for a long time to *Bowman's* membrane by a newly formed tissue and leave the cornea intact; in other cases it destroys the greater part of the cornea. In the same way the neoplasm may develop superficially on the sclera, or it may follow the lymph sheath of the anterior ciliary arteries into the eyeball early in its course. Epitheliomata of the conjunctiva may be hard or soft, and it should be mentioned that sometimes they are not flat, but beanlike, or conical.

The majority of patients with epithelioma of the conjunctiva are over forty years of age, though there are many deviations from this rule. The diagnosis is made chiefly by the exclusion of the characteristic features of the sarcoma.

When we examine a section of an epithelioma under the microscope the proliferated processes of epithelium with their branches and rounded edges can be seen, together with the epithelial pearls produced from the horny cells in their midst. Many epitheliomata start from the acinous glands of the conjunctiva. Both round celled and spindle celled sarcomas are met with, as well as tumors that contain both varieties of cells, and others with alveolar construction throughout. The rich vascularity of these sarcomas is characteristic. The pigment may be seen in the same section to be both in the tumor cells and in the cells of the intermediate tissue.

The prognosis of both of these malignant tumors need not be dwelt upon in detail. It is true that metastases from sarcomata of the conjunctiva are infrequent, though they have been met with, but the tendency to recurrence proves their malignant nature. Recurrences sometimes take place in entirely new sections of the conjunctiva and these are more apt to penetrate deeply into the eye. The epithelioma exhibits the same tendency to recur and likewise rarely gives rise to metastases. The situation of these tumors often renders radical extirpation impossible and at the same time the ophthalmologist naturally wishes to preserve the eye as long as he can; this accounts for their marked inclination to recurrence.

When either of these tumors is met with an effort should be made to remove it as expeditiously and thoroughly as may be practicable. If the eye is blind from any other cause, or if it is so seriously impaired by the growth that useful vision cannot be regained, it should be enucleated. Only in cases in which the patient refuses to allow this to be done should any attempt be made to extirpate the tumor alone. But when the eye is still capable of useful vision, and particularly when the other eye is blind, an attempt should be made to remove the growth and at the same time preserve the eye. In the performance of this operation the diseased portion of the conjunctiva is removed and the tumor dissected out of the cornea; then the wound in the cornea is cauterized with the galvanic cautery and the conjunctival gap is either covered with conjunctiva by means of a plastic operation, or filled in by a graft of mucous membrane taken from the lip. This conservative operation is of more practical use in epithelioma than in sarcoma. It is necessary that the patient remain under constant medical supervision so that intervention may be timely when a recurrence takes place. In this manner eyes have been finally saved in spite of many recurrences, as in cases reported by *Valude*, *Hartmann*, *Rogman*, *Peschel* and *Kipp*. The chances are not so good in sarcoma of the conjunctiva, for cases have been reported in which even early enucleation was unable to prevent metastasis, as in the one reported by *Kerschbaumer*. When the tumor is so large that the greater part of the conjunctiva would have to be removed with it the eye should be enucleated, because such an extensive cicatrization as would be produced by the removal of the growth would cause distortion and immobility of the eyeball. In more advanced cases, especially when the tumor has entered the eye along the anterior ciliary vessels, nothing but enucleation, or exenteration of the orbit should be considered.

22. Syphilis and Pemphigus of the Conjunctiva

A young person has noticed for three weeks a growing, painless swelling of the lower lid. When this is everted the conjunctiva palpebræ is seen to be reddened, the conjunctiva bulbi to be slightly chemotic, while from the transition fold springs forth an oblong tumor, as large as a bean, of a dirty whitish red color, with its surface ulcerated and covered with a coating of fibrin. Through the lid the growth can be felt to be hard, especially at the edges, where it has almost the consistence of cartilage. The preauricular and submaxillary lymphatic glands are swollen, hard and painless. The diagnosis in such a case a priori is not easy, for the initial lesion of syphilis is rarely met with on the conjunctiva; about fifty cases have been reported. At first there are no positive signs by which it may be distinguished from tuberculosis, or perhaps cancer, of the conjunctiva, but the diagnosis may be made by an examination for the spirochæta pallida which, when present, proves the lesion to be a chancre.

The conjunctiva may be affected in all three stages of syphilis. The relatively most frequent seat of the initial lesion is the conjunctiva of the lower lid and the lower transition fold. The infection may take place in various ways. This case, reported by *Michel*, is very instructive: A physician examined a patient who was suffering from syphilis. Afterward he thoughtlessly rubbed one of his eyes with his finger. This was followed by a slight irritation and swelling of the conjunctiva that did not improve. In a few weeks a chemosis appeared, together with a dirty yellow discoloration in the lower transition fold. Meanwhile the preauricular gland became swollen and finally the usual symptoms of secondary syphilis developed.

Sometimes the infection is conveyed by a kiss, more often by the evil custom, prevalent in many lands, of removing a foreign body from the eye with the tongue. Persons who were taking care of syphilitic children have been inoculated by them.

The therapy may be briefly stated to be an energetic antisyphilitic treatment immediately instituted. The prognosis is good; the lesion will retrogress completely without having caused injury to the eye.

Whether the obstinate catarrhal conjunctivitis occasionally met with in syphilitics is specific or not, it is certain that the conjunctiva is sometimes involved in the secondary stage of syphilis. The very marked paleness of the conjunctiva during the early part of this stage, described by *Wilbrand* and *Stœlin*, may be seen not infrequently. *Michel*, *Goldzieher* and *Sattler* have observed the development of lymph follicles from proliferations of the endothelium, which retrograded under antisyphilitic treatment. Others have met with a peculiar fatty, gelatinous appearance of the conjunctiva.

True papules, either solitary or multiple, and of various sizes, are found not uncommonly on the conjunctiva, particularly on the caruncle and the plica semilunaris. When situated near the corneal margin they somewhat resemble eczematous efflorescences. They have little inclination to break down, unless locally interfered with, and undergo involution under antisyphilitic treatment, leaving, sometimes, a brownish spot of pigment, as observed by *Gutmann.*

Finally gummata are met with in the conjunctiva. Their general color is a dull red, with yellowish gray on the top and margin. Although papules generally cause no trouble, gummata excite much irritation. They extend deeply into the tissues and changes in the iris and other parts of the eye are to be expected. They may undergo involution, but serious ulcerations are apt to take place with the production of cicatrices that resemble pterygia.

The domain of hereditary syphilis in the eye is the cornea and uveal tract; specific conjunctivitis is rarely met with in children suffering from this disease.

In pemphigus of the conjunctiva such vesicles as are to be found elsewhere on the body are rarely to be seen, because their pellicles are very thin and easily destroyed; their places are taken by sores covered by gray deposits.

According to *Michel* pemphigus attacks by choice the conjunctiva at the inner canthus and on the lower part of the globe. The disease is almost always bilateral, although the course in the two eyes may be very different. The skin and mucous membrane may be affected simultaneously, or in succession. The cicatrization that arises from these sores cannot be checked. The hair bulbs are involved in the contraction of the conjunctiva, which causes the edge of the lid to bend inward, and produces as the first results of this grave disease entropion, trichiasis and distichiasis. Ectropion occurs only in the rare cases in which the pemphigus is situated on the skin of the lid. Very often an ankyloblepharon, an adhesion of the margins of the lids, is formed at the inner commissure. Finally the entire conjunctival sac becomes obliterated, and the cicatricial, xerotic conjunctiva passes directly from the narrow palpebral fissure to the anterior surface of the eye. The cornea is spared only in exceptional cases. Infiltrates and ulcers leading to perforation may be formed; its surface may be covered by a growth that resembles pannus; it may become xerotic, or an extension of conjunctiva like a pterygium may develop over it. The worst result of all is xerophthalmos.

The prognosis is not good. An attempt may be made to improve the position of the lids by an operation for entropion. Many attempts have been made to replace the shrunken conjunctiva by grafts of mucous membrane, but they have generally proved unsuccessful. The xerotic troubles may be alleviated by the use of ointments and of milk.

23. Amyloid and Other Degenerations of the Conjunctiva

The great majority of conjunctival diseases are inflammations of bacterial, or infectious origin, but a small group remains of diseases that are known as degenerative, because of the clinical and anatomical characteristics which they display. Pinguecula and the xerosis which is the final result of contraction and faulty nutrition of the conjunctiva belong to this group.

A patient with amyloid degeneration of the conjunctiva states that his upper lid has grown thicker gradually until it has become difficult, if not impossible for him to open his eye. On examination we find the upper lid to be thick and drooping so low as to cover part of the lower lid. No changes are visible in the skin of the lid except some shining, bluish veins. Patients with this disease frequently have suffered from trachoma, which is apt to produce a certain degree of blepharoptosis that gives a sleepy appearance to the eyes, but trachoma alone does not produce so high a degree of blepharoptosis as this, or such a volumetric increase in the lids. The upper lid may be everted with some difficulty and then we see that its entire conjunctiva has a fatty, gelatinous and waxy appearance. Its surface is smooth; toward the transition folds may be seen some elevations and swellings. The bleeding produced by seizing it with forceps is very slight as compared with that caused by similar treatment of a thick, trachomatous conjunctiva. The eye exhibits few signs of irritation and the cornea is unharmed.

Clinical experience has demonstrated that amyloid degeneration of the conjunctiva is not dependent on trachoma; the connection between the two diseases in any case is accidental, for this degeneration may take place in a perfectly healthy eye. It is in no way connected with the general amyloid degeneration that results from long continued suppuration; it is a purely local disease of the conjunctiva and its cause is unknown. It is met with very rarely, appears during middle life and lasts an extremely long time. It usually starts in the transition folds, first involves the palpebral conjunctiva, and then may extend to the conjunctiva bulbi and the plica semilunaris. Sometimes it starts as a well defined tumor. Its development is always greatest in the upper lid, to which it may be confined. In the great majority of cases the cornea is spared, unless injured mechanically, or by other disease. The secretion of the eye is not increased.

This degeneration is produced by a substance which gives the amyloid reaction and stains mahogany brown with *Lugol's* solution, or greenish on the addition of sulphuric acid.

Substances that give the hyalin reaction are also occasionally met with, and cases have occurred in which hyalin degeneration was not only the more prominent, but the sole form present.

A remarkable change takes place in the vessels; their walls become homogeneously thickened and partially transformed into layers of degeneration, between which lie infiltrates containing round cells, plasma cells and giant cells. Calcifications may be found in a late stage of the disease. The whole degeneration seems to start from the walls of the vessels and the fibres of connective tissue.

Although at first glance this disease would seem to be very serious, and although its duration is so prolonged, yet the prognosis is not bad, because the cornea is usually uninjured and much improvement may be secured by operative intervention. Several sittings are necessary in which the degenerated masses of tissue are to be removed, part at a time. These partial excisions excite spontaneous resorption of the remainder of the masses.

Another and still rarer degenerative disease of the conjunctiva is the one first described by *Leber*, characterized by an enormous amount of calcification and known as *conjunctivitis petrificans*. These cases begin with an attack of conjunctivitis, in the course of which white spots appear in the mucous membrane. The epithelium over the larger of these spots is cast off, laying bare hard, chalky masses that are firmly adherent to the subjacent tissue. Many attacks follow each other and result in great cicatrization. The chalky masses are composed of lime, probably in an organic combination with a base consisting of degenerated connective tissue. The great amount of lime excreted in such cases may be appreciated from the fact that in one case, after three attacks, the total quantity removed by *Sæmisch* filled a glass of 2 ccm capacity. The cause of this degenerative disease is utterly unknown.

But these degenerative diseases are not all rarities; a very common one is that in which deposits of lime are formed in the conjunctiva. A patient with this trouble presents the cardinal symptoms of a chronic conjunctival catarrh, with little yellow deposits scattered irregularly over the tarsal conjunctiva of the upper lid, as well as in the transition fold, and elevated somewhat above its level, each usually surrounded by an area of hyperæmia. These are little *concretions of lime* in the surface of the conjunctiva. They may be either the cause or the result of the hyperæmia and inflammation. Deposits of lime may form in the *Meibomian* glands whenever the escape of their secretion is interrupted, and they establish themselves in the invaginations of epithelium which are so readily formed in chronic catarrh. Inversely, such a deposit may irritate the conjunctiva and produce the same feeling as that caused by the presence of a foreign body. Whoever does not think of this may treat a chronic conjunctivitis in vain. After the eye has been cocainized the concretion may be lifted out with a foreign body needle, or, if more deeply seated, with a small sharp spoon after a minute incision has been made down upon it.

24. Tuberculosis of the Conjunctiva

I. A Clinical Picture of Tuberculosis of the Conjunctiva and the Clinical Differential Diagnosis

Our patient, a young man, never before had any disease of his eyes. About a year ago his left eye began to tear, or lacrymate, but he paid no attention to it. After some weeks, or months, a reddish "pimple" appeared below the inner canthus in the region of the lacrymal sac, but caused no pain or other sign of inflammation. He consulted a physician and the place was treated with ointments and plaster until it broke; since then he has been unable to rid himself of the lacrymation and suppuration. For the past six months he has noticed that the eye is always and increasingly red, sensitive and yielding much secretion. At last he noticed that one place in particular looked thickened and so has sought our assistance.

In this young man, who otherwise appears to be healthy, there is an inflammatory redness of the skin in the region of the lacrymal sac, the skin itself feels thickened and there is an escape of pus from a fistula that leads from the lacrymal sac itself. It must be noticed that according to the history this condition developed gradually without having been preceded by a phlegmonous inflammation. The lower lid appears to be thickened; on eversion a large ulcer in the palpebral conjunctiva and the transition fold becomes visible. Its margins are indented, it has a dirty yellowish red color and its base is covered by white, fibrinous masses. Elsewhere there is only a great redness and swelling of the conjunctiva.

Necrosis following a burn and the ulceration attending croupous conjunctivitis produced by streptococci, which is always preceded by a severe acute inflammation, are both excluded as causes of this ulcer by the history. In pemphigus we find sores on the conjunctiva covered by a gray coating, but pemphigus of the conjunctiva is usually associated with an eruption of the same disease on the skin and its entire clinical picture is different; the sore places cicatrize, the conjunctiva is white and tense, and fresh sores are starting in other places. Deep-seated chalazia which have ruptured through the conjunctiva and have their openings surrounded by granulations present a certain, slight similarity, but this error, if made, is quickly recognized. The prominence formed by the chalazion can be felt through the skin and ordinarily other chalazia are present. No one acquainted with trachoma can ever mistake tuberculosis of the conjunctiva for it, even when the yellowish gray nodules, that often have a cauliflower appearance, are prominently in the foreground together with the papillary proliferations. The ulcerated condition of the conjunctiva will be noticed quickly. Such ulcers are also wanting in the rare *Parinaud's* conjunctivitis. Syphilitic ulcers of the conjunctiva are extremely rare and they may be excluded by a general examination, including *Wassermann's* reaction. After all these have been excluded there remains only tuberculosis of the conjunctiva. The chronic character of the disease and the unique clinical picture are both indicative of this diagnosis. The disease of the lacrymal passage in this case is most probably tuberculous also.

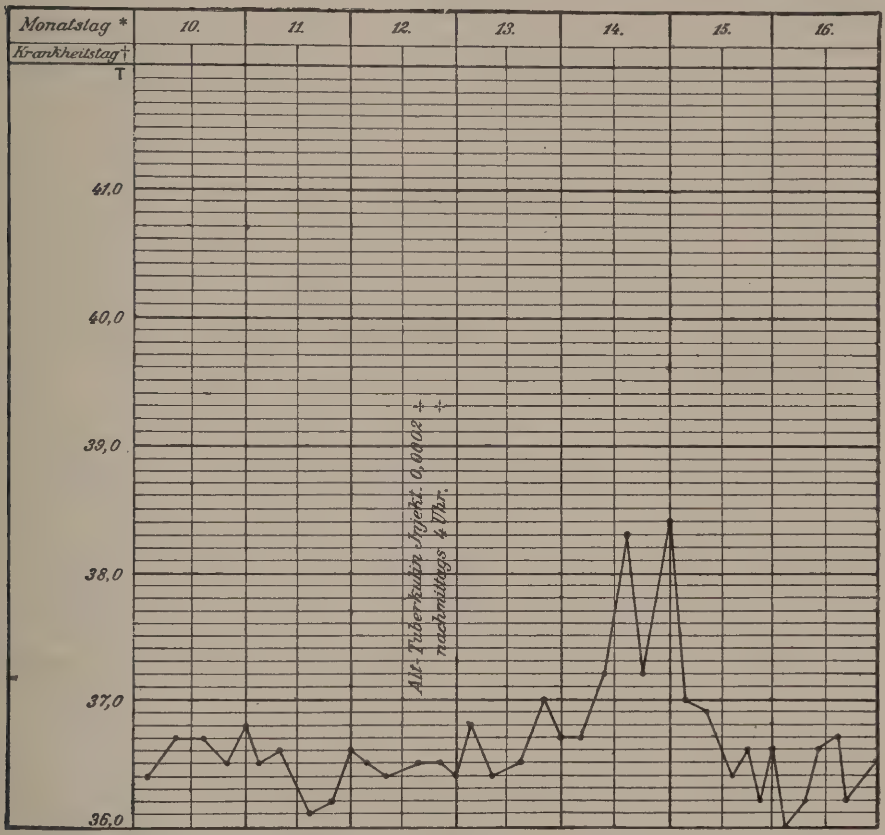
II. Procedures to Confirm the Diagnosis

A physical examination must be made of the patient for signs of organic tuberculosis, but this may not give a positive result. The bacteriological examination of slides as a rule fails to give confirmation, because the tubercle bacilli present are few in number and even when they are found the question arises whether the characteristically stained bacilli are really those of tuberculosis, or similar acid fast bacilli. The cover glass examination is, in short, of no practical importance. We cannot wait for the slow growth of tubercle bacilli in cultures and it is difficult to isolate them from the other parasites of the conjunctiva. The tubercle bacilli may be found somewhat more easily in the pus from the lacrymal sac, as *Morax* has pointed out.

A portion of the diseased tissue may be excised and examined microscopically; the bacilli are not easily found because they are few in number and if none are seen the finding of a typical tubercle with central necrosis will serve to confirm the diagnosis, but still this method is not satisfactory.

The surest proof of the presence of tubercle bacilli is obtained by inoculation in the lower animals. The eye of a rabbit is cocainized, a little puncture is made in the cornea a short distance from the limbus, the aqueous is allowed to escape and then a bit of the diseased tissue is introduced into the anterior chamber, by means of a spatula, and left lying on the iris. This inoculation is easily performed if the puncture is not made too large and the eye is held quiet during the introduction, by means of the lids. The tissue introduced must be as free as possible from other morbific agents and therefore must not be taken from a part of the ulcer that is covered by pus. If tubercle bacilli capable of producing an infection are present, a tuberculous iritis will develop in the course of two or more weeks.

The intraperitoneal inoculation of guinea pigs is still more satisfactory, as these animals are extremely sensitive to tuberculosis and larger quantities of triturated tissue can be injected. These experiments afford a positive differentiation concerning the tubercular nature of the disease, but considerable time is lost before the differentiation is made. Finally we come to the tuberculosis reactions, with the management and diagnostic importance of which we became familiar in the study of tuberculosis of the iris. *Pirquet's* cutaneous reaction may be tried first, then old tuberculin may be injected subcutaneously, when the subsequent characteristic temperature curve will leave no doubt that the patient is tuberculous, and we may confidently expect that the animal experiment will demonstrate the tuberculous nature of the lesion in the eye.



* Day of the Month. † Day of the Disease. ‡ Injection of 0.0002 of old tuberculin at 4 p.m.

FIG. 18.

III. Nature and Course of Conjunctival Tuberculosis

Although, when we consider the enormous dissemination of pulmonary tuberculosis, it is probable that tubercle bacilli come in contact with the mucous membrane of the eye very frequently, yet tuberculosis of the conjunctiva is a very rare disease. It is met with almost exclusively in young people between the ages of ten and thirty, a fact that accords with our knowledge that the susceptibility of man to the infection of tuberculosis decreases with the advance of age. Exceptionally conjunctival tuberculosis may be met with in old age.

This marked lack of receptivity to tubercle bacilli on the part of the conjunctiva has been ascribed to the fact that the lacrymal fluid is not a good nutritive medium for bacteria, that it removes the bacilli mechanically, and that the uninjured epithelium prevents the entrance of the bacilli into the tissue, but whether these factors alone are able to account for the rarity of conjunctival tuberculosis has not yet been decided.

Clinical observation has taught us that the presence of an injury favors the development of conjunctival tuberculosis. According to *Fuchs* this disease is very apt to start about the sulcus subtarsalis, a favorite place for the lodgment of little foreign bodies, and cases have been reported in which it directly followed a deep seated injury to the conjunctiva, or an operation.

The clinical picture presented by this rare disease varies in many details. *Sattler* and *Eyre* divide it into several varieties, but these merge into one another. The form most frequently met with is the tuberculous ulcer, which may be of a dirty yellow red color, be covered by a yellowish white coating, or may have a fatty appearance. Its margins are irregular in outline and indistinct, and yellowish gray tubercle nodules are to be seen in its neighborhood.

In other cases fissured papillary proliferations predominate, or, exceptionally, there may be a single, pedicled granulation tumor. In still other cases the conjunctiva is swollen diffusely and holds a great many nodules beneath its epithelium, the miliary form, or the diseased part may have the appearance of a large, spongy granulation.

The cornea is not involved in some cases, but it is very apt to be affected in a variety of ways. A sort of pannus, in which nodular elevations are visible, may be formed over it, or it may be the seat of extremely obstinate ulcerations, or thick tuberculous granulations may proliferate over its marginal portions from the conjunctiva and eventually cause necrosis of the cornea.

These variations in the clinical picture of conjunctival tuberculosis correspond exactly with the behavior of the disease in other organs, and are due to differences in the susceptibility of the patients, in the virulence and quantity of the tubercle bacilli, and in the reactions of the diseased tissues. The same causes explain also why the course varies so greatly in different cases.

The prognosis of this disease as regards the eye is very grave. The infiltration may extend from the conjunctiva to the tarsus, the ulcers may destroy gradually the transition folds, the conjunctiva bulbi, the cornea, the sclera and even the eyeball itself. Extensions of the disease to the skin of the lid have been observed. All of these complications are especially likely to occur when the course of the disease is attended by severe ulcerations, particularly when the slight troubles that first appear have not received the timely treatment by which they can usually be checked, even after the cornea has become involved.

Relapses may be anticipated for a long time.

Benign forms that cicatrized and healed spontaneously have been met with, but it must be borne in mind that such a focus of tuberculous inflammation in an eye is a menace to the entire body and may lead to general infection. Cases have been observed in which a man previously free from tuberculous taint was attacked by tuberculosis of the conjunctiva, followed by tuberculosis of the lacrymal passages, of the nose and finally of the lungs, which proved fatal, and such cases prove that a general infection can take place in man from the conjunctival sac, as it has been proved experimentally by myself and others to take place in animals.

However the clinical picture of conjunctival tuberculosis may vary in individual cases, the pathology always remains the same. Tuberculous nodules develop in the thickened and greatly infiltrated conjunctiva, which is undergoing papillary proliferation, that may be recognized readily on the slide through their giant cells. They present the appearance of vascular granulation tissue containing giant cells surrounded by epitheloid cells. Tubercle bacilli are not always to be found easily in the sections, they are generally few in number. The tendency to necrosis varies much. Caseation begins in the protoplasm of the giant cells and the necrotic tissue may be seen under the microscope to be surrounded by the exuberant granulation tissue. The entire tissue breaks down and the tuberculous ulcer is formed. The proliferated papillary bodies become great, tuftlike growths. The processes of cicatrization vary greatly.

Although this disease usually starts in the conjunctiva tarsi it may occasionally begin in the conjunctiva bulbi.

IV. Origin of Conjunctival Tuberculosis

Two possibilities are a priori present: the tubercle bacilli may be carried into the mucous membrane by metastasis, or they may be introduced from external sources.

In such a case as this the patient should be examined first for signs of tuberculosis in other organs, particularly the lungs, joints and glands, in addition to the lesion in the conjunctiva. When other organs are found to be diseased metastasis cannot be excluded with certainty in all cases, especially when the affection is bilateral, when several isolated patches can be seen in the mucous membrane, and when the disease presents the miliary form. Investigators are inclined today to ascribe a hæmatogenous origin even to lupus.

But the origin of the infection in a case of conjunctival tuberculosis may be ectogenous, even when the patient has other tuberculous lesions. The infection may be propagated directly to the conjunctiva from a tuberculous disease of the lacrymal sac. Bear in mind that the dacryocystitis which accompanies lupus of the face is usually, at least at first, a simple, purulent, nonspecific disease. It is also well known that lupus of the skin rarely extends over the margin of the lid to the conjunctiva, but on the contrary is accustomed to leave that margin intact. Yet in the individual case a direct transmission must still be thought of. It is not surprising that the conjunctiva finally becomes infected when tubercle bacilli are brought to it continually in the pus from a diseased lacrymal sac.

In the presence of other tuberculous diseases ectogenous infection of the conjunctiva may be produced in various ways. It may be carried by the finger from a lupus. It may not seem plausible that a conjunctival tuberculosis, which begins beneath the upper lid without a preceding injury, should be ascribed to an ectogenous infection, but when the tubercle bacilli are in the conjunctival sac and the necessary susceptibility is present, who can exclude a rubbing of the eye after the lapse of so long a time?

Patients with pulmonary tuberculosis can infect their eyes with tubercle bacilli, and it is strange that this does not happen more often, because the sputum frequently is brought into contact with the eye by means of the hands, or handkerchiefs.

It has been established clinically that in the majority of cases no other signs of the disease can be found, and that it is therefore a local tuberculosis. Hence conjunctival tuberculosis is usually due to an ectogenous infection, although it can scarcely be determined in any particular case in which way the disease originated.

V. Treatment of Conjunctival Tuberculosis

These cases demand hospital treatment. An attempt must be made first to remove the tuberculosis in the lacrymal sac. The nature of the operative procedure for this purpose depends on the condition in the nose; if the bones are involved a cure is rendered extremely difficult. Conservative treatment with antiseptics is useful in the conjunctiva only in benign forms of slight extent.

The diseased tissue is to be carefully removed and curetted away, and the surface of the wound is then to be cauterized with the galvanic cautery. This should be done in several sittings, in order to observe how the eye bears such operative treatment. Cauterization of the sores with concentrated, or 50% lactic acid has been recommended. After the pathological focus has been removed in this way the loss of great portions of the conjunctiva, together with the attendant cicatrization, may result in such sequelæ as distortion of the lids and symblepharon, which must be treated later.

Tuberculin treatment of the entire body may be of assistance, but alone it is incompetent to deal with conjunctival tuberculosis.

Lepra is met with in the eye in both the tuberous and the maculo anæsthetic forms. In the tuberous form the nodes begin by predilection in the eyebrows.

25. Diseases of the Plica Semilunaris and Caruncle

An easily movable, reddish gray tumor, about as large as a pea, can be seen in the inner canthus of this young patient, and is still visible between the margins of the lids when the eye is closed. After the eye has been cocainized this tumor may be seized with forceps and lifted up, when it will be seen to be over the caruncle, to be attached thereto by a pedicle, and to bleed freely. The caruncle is a residual formation of cuticle and may form the source of origin of all forms of benign and malignant growths met with in this region, although the cases are rare. The polypoid tumors, the papilloma and fibroma are the most common of the benign forms. In the present case the tumor is probably a soft fibroma, the hard variety does not bleed so readily. We have already learned that angiomata of the conjunctiva may start in the caruncle and the other benign tumors need only to be named, the nævus, dermoid, lipodermoid and cyst, they have already received consideration. The malignant tumors, sarcoma and carcinoma, which are usually situated in the conjunctiva near the limbus, occasionally start in the region of the caruncle. The sarcomas are usually pigmented and sometimes commence in a congenital nævus. The clinical symptom common to all these neoplasms is that they contract the space at the inner canthus, and interfere with the formation of the lacus lacrimalis and with the outflow of the tears. The treatment is purely surgical; such a polyp as this is removed and its base destroyed with the galvanic cautery.

Aside from these neoplasms the caruncle sometimes displays congenital anomalies of form. It may be partially or wholly absent, as in microphthalmos and anophthalmos, sometimes it is very small, sometimes quite hypertrophic, and duplicatures have been observed occasionally. The plica semilunaris lies in immediate proximity to the conjunctiva and takes part in many inflammations of that membrane to the extent of becoming reddened; more rarely it becomes inflamed itself. *Eversbusch* and others have observed it to be the seat of syphilitic thickening, as well as of the primary lesion and of the papules of syphilis. The plica can also hypertrophy spontaneously, so as to extend to the margin of the cornea and to resemble the nictitating membrane of many animals. It is of more practical interest to know that foreign bodies may lodge beneath it and cause it to swell; this is to be remembered when an eye is examined in search of a foreign body. A lively irritation of the plica semilunaris is excited more frequently by the rubbing against it of an eyelash caught in and protruding from the lower canaliculus.

26. Clinical Picture and Course of Conjunctivitis Trachomatosa

In order to understand the clinical pictures of this disease it is necessary to begin with its fundamental characteristics.

Trachoma, also known as conjunctivitis granulosa, is a wholly specific, infectious disease of the human conjunctiva, the nature of which is to develop a chronic, inflammatory hypertrophy of the mucous membrane and to transform the portions of conjunctiva affected into cicatricial tissue.

This hypertrophy of the conjunctiva, which is brought about by a diffuse inflammatory infiltration of its adenoid layer, manifests itself in two different ways; in a proliferation of the papillary bodies, and in the development of trachoma granules. These give the conjunctiva a rough, or granular appearance, whence the disease has derived its name (τραχυς, rough). In most cases these two forms of hypertrophy are associated and no separation of the "papillary" and "coarsely granular" varieties can be made, both appertain to the same disease. But the name trachoma should be applied only to such diseases of the conjunctiva as are able to inflict permanent injuries upon the eye, to those only in which the hypertrophy of the conjunctiva ends in the formation of cicatricial tissue, and such a differentiation is by no means always easy. First we will study the typical picture of trachoma at the height of its development.

I. Demonstration of Cases in Early Stages of Trachoma

Case 1. Complete Picture of Granular Trachoma

The eyelashes are glued together in bunches by a tenacious, mucous secretion which can also be seen dried on the margins of the lids and at the canthi. The patient says that the lids are stuck together in the morning and can be opened only with difficulty. This secretion of the trachomatous conjunctiva is of importance in the origin and spread of the disease.

The eye is irritated, the patient blinks constantly and complains of photophobia, as well as of a feeling of pressure and burning in the eye.

Although no changes are to be seen in the skin both upper lids appear to be voluminous; they droop over the cornea, evidently move with some difficulty and consequently the palpebral fissure is not as wide open as normal. The thickening of the upper lid is greater toward the margin of the orbit. When the lower lid is examined carefully a slight increase of volume can be detected, but it is much less than in the upper one. The conjunctiva scleræ is moderately injected, especially in the posterior vessels, and the plica semilunaris is somewhat red and swollen. The characteristic changes of trachoma are visible on the conjunctiva tarsi and transition fold as soon as the upper lid is everted. The lid is not everted as easily as in a normal eye and the difficulty encountered suggests to the expert the condition he may expect to find.

The upper transition fold is enormously swollen and protrudes, presenting several thick folds. It contains rows of granules not evenly arranged, that extend nearly from one canthus to the other. These granules are for the most part roundish, some hemispherical, some flattened, many fungoid; they are smooth, reddish gray in color, and shine through the membrane. The general appearance resembles that of cooked grains of sago, or of frog spawn. The size of the individual granules varies from 1 to 2 mm in diameter. Analogous prominences are to be seen at the place where the transition fold borders on the tarsal conjunctiva. Both here and in the transition fold the mucous membrane is arched upward by the trachoma granules.

If this swollen transition fold is pinched between the fingers a gray, gelatinous mass escapes from some of the granules which then become flattened. The nearer the granules approach the conjunctiva tarsi the more plainly apparent is a change in their character.

Granules are extremely numerous throughout the conjunctiva tarsi, but they are much smaller than in the transition folds and are not elevated above the surface of the mucous membrane, through which they glisten as little, round, yellowish, or whitish red spots. This altered demeanor of the granules is due to the fact that the conjunctiva over the tarsus is firmly adherent to the subjacent tissue and therefore cannot be forced up so readily. In the later stages of the disease these granules do become slightly elevated. Analogous changes are present in the lower transition fold and the conjunctiva of the lower lid, but the granules are much less developed. This fact is of diagnostic value: the disease usually begins in the conjunctiva of the upper lid and of the upper transition fold, and is always developed to the greatest degree in that locality. It should be mentioned that the formation of granules may pass from the transition fold into the conjunctiva scleræ and even into the plica semilunaris, in which sometimes it may start.

Case 2. Preponderance of Papillary Hypertrophy

We have learned to recognize one form of trachomatous conjunctivitis, but we must be on the watch constantly for another. If you will look at the mucous membrane in the immediate neighborhood of the granules in this case, you will see that they lie in a conjunctiva which presents a peculiar velvety appearance. This velvety appearance is caused by a swelling and infiltration of the papillary bodies, together with an inflammatory infiltration of the adenoid layer. The conjunctiva is rough, not from the granules, but in consequence of the development of the papillæ. Numerous fine, cylindric elevations, closely packed together, are to be seen about the granules, especially at the junction of the conjunctiva of the lid with that of the transition fold; some of them are so large that they surround the granules like little warts and make the surface of the membrane very rough. Those in the conjunctiva tarsi are much finer and produce its velvety appearance.

Case 3. Thickening of the Tarsus

In our next case, in which the other changes are analogous to those just mentioned, the thickening is no longer confined to the conjunctiva, but has invaded the tarsus. The upper lid is everted with greater difficulty than before, so that the expert knows at once that the tarsus is involved and that contraction is to be feared. When the lid has been everted its upper part is seen to be thickened and protuberant, as well as the transition fold, because the tarsus and subtarsal tissue have become greatly infiltrated.

Case 4. Well marked Pannus

Hitherto we have dealt with cases in which the pathological changes were confined to the conjunctiva. Our next case presents that which makes trachoma such a pernicious disease, the trachomatous disease of the cornea, pannus trachomatosus. The upper half of the cornea is covered by a reddish gray cloud that contains numerous superficial, ramifying vessels. The surface of the cornea is slightly wavy. It is characteristic of pannus that it comes from above and is separated from the healthy cornea by a sharp line of demarcation. The eye thus affected suffers great irritation. The papillary hypertrophy and granules are analogous to those in the previous cases.

It can be readily understood that the roughened conjunctiva of the upper lid may irritate mechanically the surface of the cornea, and it must not be forgotten that the upper portion of the cornea is in direct contact with the diseased tissue, especially when the eye is closed. Thus abundant opportunity is given for a contact infection, provided that the morbific agent of trachoma can get a foothold in the cornea.

II. Later Stages of Trachoma

It was stated at the beginning of this lecture that the nature of trachoma differs from that of most other infectious forms of conjunctivitis in that no restitutio ad integrum takes place, but that a cicatricial contraction follows the hypertrophy of the mucous membrane. A few cases will illustrate the stages of this change.

Case 5. Formation of Cicatrices in the Conjunctiva Tarsi

The first cicatricial stage is presented in this patient. The conjunctiva tarsi is cut up into islands of thickened tissue by narrow, whitish, cicatricial bands. One band is somewhat broader than the others and runs almost the entire length of the lid parallel to and a few millimetres distant from its edge. Granules may still be found in some cases, but not in others. The picture may be very different in the two eyes. Relapses, in which the formation of granules is increased again, occur not infrequently and as a rule the pannus grows worse when these occur in the cicatricial stage. Small, fresh infiltrations appear between the vessels in the pannus, or ulcers are formed which persist for a long time and give rise to severe subjective symptoms.

Case 6. Gelatinous Trachoma

Here is an old case of trachoma which presents a very different picture. The thick swelling of the transition fold looks peculiarly glassy, or gelatinous. The follicles have become confluent and granules can scarcely be found. At first the reddish gray tone of the fresh granules, or the frog spawn appearance of the older ones, disappears and the follicles assume a more faded gray tone, until they are finally submerged in the peculiar gelatinous tissue. These are the signs of a grave, regressive metamorphosis of the trachomatous conjunctiva.

Case 7. Trichiasis

The next patient exhibits a more advanced stage of the cicatricial contraction. Even from a distance it can be seen that the upper lid is no longer normal. When it is lifted up a little the free margin can be seen to be directed inward.

The inner edge is rounded off and the lashes are directed not forward, but downward and even inward, so that some of them rub against the cornea with every movement of the lid. The distorted cilia appear to be stunted and thin. The eye is in a condition of great irritation and a pannus has been formed on the cornea as the result of the constant scouring of the lashes. The cause of this condition is revealed when the lid is everted, a manœuvre that is now so difficult that the assistance of a glass rod is needed. The conjunctiva tarsi is seen to be crossed by many intersecting cicatricial bands, the largest lying in the sulcus subtarsalis at the margin of the lid. The upper transition fold still protrudes, but as a smooth gelatinous swelling, not fissured and granular as before. Cicatrization has taken place. The shrivelled conjunctiva tarsi is contracted and the tarsus, to which it is firmly adherent, has lost part of its elasticity and is itself infiltrated and thickened. It is at first tensely curved like a bow and finally becomes bent at an angle. This condition of the tarsus, together with the rounding off of the inner edge of the margin of the lid, causes the lashes to take a more and more oblique position, and produces trichiasis.

Case 8. Entropion

Finally comes the worst case of all, a case of well marked entropion. The palpebral fissure is shortened, the upper lid is arched, the cilia have almost disappeared. When the lids are separated the lashes can scarcely be seized, they are so stunted. The inner edge of the margin of the lid is almost entirely gone, so that the skin seems to pass directly over into the conjunctiva. The lid can be everted only with very great difficulty and then a deep troughlike concavity and contraction of the lid comes into view. A white, smooth scar has taken the place of the entire conjunctiva tarsi and the transition fold is completely gone. When the lower lid is drawn down, vertical, cicatricial cords are to be seen stretching across directly to the eyeball, a condition known as symblepharon. The cornea is covered everywhere by thick, vascular opacities. This case demonstrates the completed transformation of the conjunctiva into cicatricial tissue, the contraction and abnormal curvature of the tarsus, the shortening of the palpebral fissure, the constriction of the entire conjunctival sac; it is a case of entropion with trichiasis and distichiasis.

III. The Most Important Points in the Course of Trachoma

These eight cases illustrate the most common and most important clinical pictures of trachoma with which we have to deal. From the standpoint of the patient it is a very serious disease and I shall later deal with its general and social importance in civilized countries. At present the most important points in its course may be briefly elaborated.

Trachoma rarely begins as an acute inflammation. In certain cases a sudden inundation of the eye with the morbific agents may perhaps occasion a tempestuous commencement of the disease in the form of a severe catarrh, but such a case is exceptional. It is characteristic of trachoma that it begins insidiously and follows a chronic course. It begins so insidiously that months and even years may elapse before the patient becomes aware of the presence of disease. This is the more dangerous because these persons can spread the contagion and infect other people.

In countries, like Egypt, in which trachoma is endemic, many trachoma patients are met with suffering from violent catarrhal inflammation, but the great majority have been found to be cases of mixed infection in which *Koch-Weeks* bacilli, or gonococci, were present. In our cases the beginning is nearly always chronic.

Almost imperceptibly, or with only very slight catarrhal symptoms, the mucous membrane becomes rough and uneven in consequence of the hypertrophy of the papillæ, between which the granules gradually shine through more and more clearly. These granules develop first and to the greatest degree in the upper transition fold.

Both mild and severe cases are met with, as might be expected; this is convincingly demonstrated by the results of treatment, but the disease generally displays little inclination in any way to a rapid, spontaneous cure. The development of the granules increases, they lie in rows in the transition fold, crowd forward its protrusion more and more and gradually invade the conjunctiva tarsi.

This invasion of the tarsus is one of the most serious symptoms. The inflammatory infiltration is most marked at its lower margin where it is perforated by vessels passing to the conjunctiva along the line of the sulcus tarsei inferior. The infiltration passes along these vessels to invade the tarsus.

These changes cause the upper lids to droop and produce the blepharoptosis characteristic of trachoma. The thickened lids naturally press more and more upon the eye and increase its irritation and secretion, but both of the latter depend in a great degree on the vocations of the patients. Many weeks and even months may elapse before this stage is reached. The condition becomes bad when the cornea finally becomes affected.

The great danger of trachoma lies in the involvement of the cornea. This complication begins as a swelling of the upper limbus with a distinct pericorneal injection. Then minute, scarcely visible infiltrates appear very superficially in the cornea near the limbus and the first little vascular sprouts shoot forth into the cornea from the superficial marginal network of vessels. At the same time the pupil becomes contracted and reacts to light slowly, while the eyes become photophobic. If fluorescin is dropped on the cornea in such an early stage little green spots can be seen frequently in the epithelium; they are the precursors of a pannus. At this stage the corneal disease may clear up completely.

In other cases new vessels continue to form and to ramify, new infiltrations appear between them, opacities form and blend together and the surface of the cornea becomes uneven, producing again the picture of trachomatous pannus, which may last a long time on the upper part of the cornea and presents a sharp line of demarcation.

The word pannus means cloth. According to *Hirschberg* the Arabs called the zone of the cornea covered by pannus *sebel*. When this granulation tissue is thin and contains few vessels it is called pannus tenuis; in this condition pannus is capable of undergoing an extensive involution. But in many cases the pannus involves the parenchyma and is raised distinctly above the level of the cornea, when it is known as pannus crassus. Such a pannus is developed when the lashes are bent inward and are caused to rub against the cornea in consequence of an abnormal curvature of the lid.

In very bad cases the entire cornea is covered by pannus, in which all the vessels do not come from above, but some emerge from below and from each side to spread over the cornea.

It is of considerable clinical importance to know that pannus may begin in any stage of the disease, its development depends in no way on the degree of the conjunctival disturbance. *Kuhnt* maintains that the condition of general nutrition exercises some influence over its production, but it is self evident that the occurrence of this contact infection is determined by the number and nature of the morbific agents which act upon the epithelium of the cornea. Pannus appears in more than 50% of the cases of trachoma that are not treated. During the course of a trachoma oval ulcers may be formed in the cornea which are characteristically situated at the margin of the pannus.

Severe pannus and ulceration of the cornea is often associated with an obstinate iritis, which is probably caused by the entrance into the eye of metabolic products from the trachomatous cornea, and their action upon the iris.

The danger from trachoma inheres in the visual disturbances that such troubles of the cornea are bound to produce. All of these processes are extremely slow.

The clinical picture changes gradually as the cicatrization and its resultant sequelæ come more and more into the foreground. The conjunctiva gradually loses its roughness and becomes smoother. Red islands of hypertrophic tissue exist for a long time upon the tarsus between the cicatricial bands, until finally the conjunctiva tarsi has become thin and smooth. In the transition folds there are no white, cicatricial bands, but the thick swelling of the fornix becomes flatter and flatter and the conjunctiva is shortened until the transition fold has disappeared.

Sequelæ of the Cicatricial Contraction

If such eyes are not given proper and timely treatment the process of contraction advances and quickly produces its results. A series of characteristic sequelæ develop quite regularly. The upper lid assumes a characteristic bulging form as the palpebral fissure becomes shortened. The cicatricial contraction of the conjunctiva causes the tarsus to become more convex forward, to become "trough shaped," and finally to be bent at an angle. This angular bend of the tarsus and the corresponding groove in the conjunctiva are usually to be seen about 3 mm above the free margin of the lid, at the place where the sulcus subtarsalis was formerly situated.

The margin of the lid is carried more and more inward by this change in the tarsus, and its inner edge, which under normal conditions is perfectly sharp, becomes rounded off, partly by the tension of the contracting conjunctiva tarsi, partly by the pressure on the eyeball. This change in the margin of the lid is naturally accompanied by an alteration in the position of the lashes; they are directed downward and backward, instead of forward, and begin to rub against the cornea with each movement of the lid. This increases the irritation of the eye and the thickness of the pannus. This trichiasis is frequently accompanied by distichiasis.

Entropion is simply another link in this chain of misfortunes, in which the contraction has become further advanced and the entire margin of the lid has been turned backward.

The lower lid may be forced away from the eye by the hypertrophic conjunctiva and may then become entirely everted by a spasmodic contraction of the orbicularis.

The granulation tissue in the pannus undergoes a transformation into a thin, opaque connective tissue which is incapable of further involution. In other cases, in which the pannus is very thick and involves the parenchyma, the cornea may soften to a certain degree and give way before the intraocular pressure, so that various forms of ectasiæ are permitted to develop. Ectasiæ may also be produced after ulcers.

Hand in hand with its transformation into cicatricial tissue the conjunctiva loses its ability to secrete and the lacrymal secretion becomes diminished. This leads to the saddest ending of trachoma, xerosis conjunctivæ. The patients complain of an increased feeling of dryness; peculiar dry places that look as if they were smeared with fat appear on the conjunctiva and spread continually until the condition of xerophthalmos is produced. In such an eye the cornea has become completely opaque, while the epithelium over the pannus is thick, quite dry and resembles epidermis.

Although trachoma is a specific disease of the conjunctiva, it is not rigidly confined to this membrane. Every ophthalmologist, who is accustomed to treat great numbers of trachoma patients, knows that dacryocystitis is very common among them in both the early and late stages. The morbific agents may be carried by the tears into the nose and trachomatous changes have been found frequently in the wall of the lacrymal sac and in the mucous membrane of the nose. In exceptional cases trachoma has been known to be present in the lacrymal sac while not in the conjunctiva. This is a formidable disease, which demands thoroughgoing prophylactic and therapeutic measures.

27. Etiology, Differential Diagnosis and Pathology of Trachoma

The demand of ophthalmic science, that only such granular diseases as are able to permanently injure the eye shall be called trachoma, is not always to be complied with easily and with certainty, especially in the diagnosis of mild cases at their commencement. The reason for this is simple. The formation of follicles in the conjunctiva is induced by many causes besides the presence of the morbific agent of trachoma, and in doubtful cases the diagnosis of trachoma can be pronounced positive only when we are able to demonstrate the presence of the morbific agent. Unfortunately this cannot yet be done, because the identity of the agent is still unknown, though for many years it has been unceasingly searched for by investigators.

I. Etiology of Trachoma

We know that trachoma is an infectious disease from its appearance in epidemics and its spread among isolated bodies of people and families, when a member has been attacked. In spite of all care a physician may infect his own eyes; *Cuignet*, the discoverer of skiascopy, lost an eye through trachoma.

We know also that the disease is transmitted by the secretion from the trachomatous conjunctiva, which must therefore contain the morbific agent. Hence the cases that have the largest amount of secretion are the most dangerous to the persons about them. The disease is usually carried from one person to another on hands, or on articles in common use, like washbowls, or towels, which are soiled with the trachomatous secretion. It is never transmitted through the air.

Many patients with trachoma suffer at the same time from other forms of conjunctivitis, produced by other pathogenic agents. In this region the blepharoconjunctivitis produced by diplobacilli and the acute catarrhs caused by pneumococci are met with most frequently, while in Egypt gonorrhœa and the acute inflammation produced by the *Koch-Weeks*' bacillus form the usual combinations with trachoma. These secondary infections are met with so frequently in that country that some ophthalmologists have inferred a direct transition from gonorrhœa into trachoma.

In the third place we know that trachoma can be communicated to apes. *Hess* and I were the first to perform this experiment successfully. If secretion taken from the conjunctiva of a patient with trachoma is placed on the conjunctiva of an anthropoid ape a form of disease is produced which corresponds exactly with trachoma in the human eye. Follicles appear, an inflammatory hypertrophy of the conjunctiva is formed, and the microscopic examination of sections of the tissue demonstrate the existence of trachoma. The disease takes a milder course than in man, evidently because the virus is weakened, and it is doubtful if an extensive contraction can be produced in the conjunctiva of an ape. In other respects we know nothing as yet about the morbific agent of trachoma.

Recently a gentle ray of hope has somewhat illumined the darkness. Peculiar cell inclusions were discovered by *v. Provaczek* and *Halberstædter* in the epithelial cells of the conjunctiva of persons suffering from trachoma, and were thought by them to be parasitic and to be the morbific agents of this disease. *Greeff* named them trachoma bodies, and also thought them the long sought for agents of trachoma until *Heymann*, a student of *R. Pfeiffer*, demonstrated that these peculiar cell formations were present in the epithelial cells of the conjunctiva during a blennorrhœa. The relation these inclusions bear to the etiology of trachoma is undergoing a lively investigation today. The impression thus far is that these cell inclusions are manifestations of a hitherto unknown virus, which is met with in the genital tract and in the conjunctiva of man, and can be communicated to apes, in whose mucous membrane it is able to excite a follicular inflammation.

II. Differential Diagnosis of Trachoma

The diagnosis of trachoma occasionally presents a very serious question to the physician and its differentiation must be discussed rather critically, especially as, in view of this unsatisfactory situation, we have to rely on the clinical symptoms. An epidemic outbreak of an inflammation of the eye which was at first regarded as trachoma, but proved later to be a harmless conjunctivitis with a different etiology, has occurred repeatedly in schools, institutions, barracks and prisons. To guard against such errors everything that can be considered differentially diagnostic should be taken into account.

The least difficulty in differential diagnosis is encountered when the case is one of trachoma in the cicatricial stage. Any granular disease of the conjunctiva that results in cicatrization is trachoma. The diagnosis is equally positive when a granular disease of the conjunctiva is associated with a characteristic pannus, even though no cicatrization has yet taken place. It is also advisable to think of trachoma first in any case of bilateral curvature of the upper lids with pannus. To be sure other conditions exist that lead to cicatrization in the eye.

Cicatrices of the worst variety are produced in the conjunctiva by pemphigus, but this disease is very rare and the cicatrices are preceded by large ulcers covered with pseudomembranes, which are not met with in trachoma. The history and the physical examination will direct the attention of the physician to eruptions of pemphigus elsewhere on the body.

Cicatrices in the eye from burns are more common. The history is usually sufficient to determine the diagnosis, but it may be noted that in such accidents only one eye, and by preference the lower lid, is ordinarily affected.

In vernal catarrh the conjunctiva may appear to be roughened by the enlarged papillæ when the lid is everted, but the proliferations are flat, like paving stones, while the entire conjunctiva presents a diffuse, milky appearance, very different from that produced in trachoma by the cicatricial bands. The distortion of the lids, the trachoma granules and the pannus are all absent in vernal catarrh, while the peculiar hyaline proliferations at the limbus are present.

The other diseases in which cicatrization takes place in the conjunctiva may be rapidly excluded. Cases of diphtheria that leave scars in the conjunctiva are scarcely to be met with, and even after gonorrhœa serious scars are very rare. Slight cicatrices may be found in the transition folds, but not in the conjunctiva tarsi, a condition diametrically opposite to that met with in trachoma.

In neglected cases of entropion of the lower lid due to a simple chronic blepharoconjunctivitis there is a certain degree of cicatrization in the conjunctiva immediately adjacent to the margin of the lid, but it bears no resemblance to the cicatrization of trachoma. Granules are absent, but the conjunctiva has a velvety appearance due to the swelling of the papillæ.

Scarcely any difficulty attends the diagnosis of cicatricial trachoma, but a suspicious pannus demands rather more attention. In trachoma the pannus comes from above and terminates in a horizontal line of demarcation at, or just below the centre of the cornea, while cilia that have been given an abnormal direction by some other cause, such as a lime burn, may create a pannus by their friction.

Eczematous pannus is the first disease to be excluded in suspicious cases. The characteristics that distinguish it differentially from trachomatous pannus are 1, its occurrence usually in children, in adults only as a relapse; 2, the patients so afflicted are subject to scrofulosis and eczema; 3, its frequent association in the eye with eczematous efflorescences, or ulcers; 4, the conjunctiva is never granular or cicatricial, but smooth, or a little velvety in cases of severe chronic catarrh; 5, its situation may be in any part of the cornea. The last is the most important differential characteristic for, although eczematous pannus is occasionally localized in the upper part of the cornea, it starts irregularly from any point. It is not uncommon for trachoma to be combined with eczematous diseases of the eye, for scrofulous persons may become infected as well as others, and then the differentiation must be made through the results of treatment. Under such circumstances the diagnosis of a mild case of trachoma in an early stage is quite difficult.

In spite of all the assertions of various authors it seems doubtful to me whether there exists a true, acute trachoma. The occurrence of an acute trachoma cannot be denied on theoretical grounds, as long as the morbific agent cannot be recognized and demonstrated easily, for there is no doubt that these agents differ in their virulence, and it can be imagined readily that when an eye is inundated with such malignant germs an infection may take place with tempestuous symptoms of inflammation. Hence there are authors who recognize a clinical picture of conjunctivitis granulosa acuta.

At first the clinical symptoms are those of an acute conjunctival catarrh. Some weeks later granules become visible in the hypertrophic transition folds and characteristic changes take place in the cornea; vascularization from the upper marginal network and very fine superficial infiltrations in the upper marginal segment. These symptoms gradually merge into the clinical picture of chronic trachoma. I have not yet seen an unquestionable case of acute trachoma, in all cases that seemed to be such the bacteriological examination has demonstrated the presence of a secondary infection. The various morbific agents of acute conjunctivitis produce clinical pictures that resemble that of acute trachoma, and they can establish themselves in a trachomatous conjunctiva, so in order to differentiate an acute trachoma such inflammations must be excluded as gonococcal conjunctivitis, the conjunctivitis produced by *Koch-Weeks* bacilli, pneumococcal conjunctivitis and, in children, the scrofulous, so called congestive catarrh. The diagnosis of acute trachoma should not be made without a thorough, preliminary, bacteriological examination. While dealing with these suspicious cases it may be well to mention that grave clinical pictures may be produced by acute traumatisms, such as those excited by 1, beards of grain beneath the upper lid; 2, irritating powders, including the severe form of conjunctivitis excited by caterpillar hairs; 3, intentional attempts to simulate the picture of acute trachoma by the introduction of snuff, or horse powder, into the eyes.

Differential Diagnosis of Chronic Trachoma

The differential diagnosis of a granular affection of the conjunctiva, which begins and pursues its course as a chronic disease and in which the characteristic cicatrization has not begun at the time of examination, is far more difficult. We must consider under what conditions follicles are formed in the eye and how trachoma can be differentiated from the harmless follicular diseases that so closely resemble it.

The numerous follicular diseases of the conjunctiva that are benign, i.e., that retrogress without the formation of cicatricial tissue, may be divided into two groups.

When a large number of people with otherwise healthy eyes are examined quite a few will be found to have single follicles here and there in the transition folds, particularly among the children and among those who work a good deal in artificial light, or in dusty places. This condition is known as conjunctivitis sicca. There is no secretion from the conjunctiva which is only slightly reddened and presents a little papillary hypertrophy at the upper margin of the tarsus with individual follicles near the inner and outer canthi. The prolonged instillation of atropin also excites a marked development of follicles in some persons. All of these cases are benign, are not infectious and do not transform the conjunctiva into cicatricial tissue. They are not cases of trachoma.

On the other hand we meet with benign, but communicable forms of conjunctivitis in which follicles are formed. Follicles are to be found in acute pneumococcal conjunctivitis, and little epidemics of benign follicular conjunctivitis have been observed frequently in institutions. In such cases diagnosis is difficult. It has been suggested that such diseases may be distinguished from trachoma by the presence of the follicles in the lower transition fold alone, but this is not correct, because a careful examination will reveal follicles in the angles of the upper transition fold as well. It is more correct to say that in the benign forms of disease the follicles are smaller and do not invade the tarsal conjunctiva. But this difference is not decisive and we must be content in many cases with a diagnosis of "trachoma (?)." The subsequent course and the results of treatment will differentiate the benign forms because they readily get well.

A few words concerning those forms of conjunctivitis characterized by a papillary hypertrophy which need to be differentiated from the form of trachoma in which the hypertrophy of the papillary bodies predominates. In vernal catarrh there is a certain degree of roughness of the conjunctiva, but the latter has a milky, blue appearance which is not present in trachoma, while the proliferations at the limbus are distinctive. The papillary hypertrophy in neglected cases of chronic blepharoconjunctivitis with ectropion is distinguished from that of trachoma by the absence of follicles. The papillary hypertrophy which follows gonorrhœa soon passes away and no follicles are present. The rarer diseases of the conjunctiva, such as tuberculosis and *Parinaud's* conjunctivitis, do not come into consideration.

III. Pathology of Trachoma

That the adenoid layer of the conjunctiva is the principal seat of the trachomatous disease is proved clinically by the fact that the eruption of granules, which marks its commencement, takes place within it. When we look at a section of trachoma tissue these granules attract the eye and deserve attention first.

1. Sections from Fresh Cases of Trachoma

A. Anatomy of the Granule

The granules lie in part just beneath the epithelium, in part more deeply embedded in the adenoid tissue.

It can be seen at a glance that these granules are spherical aggregations of cells, and the generally accepted view today is that they are little lymph follicles. The marginal zone of the follicle is differentiated from the centre by the stain. The cells in the marginal zone are arranged with a certain regularity, sometimes like a string of pearls, about the central portion. The cells are small, round, with strongly stained nuclei and are nothing else than lymphocytes.

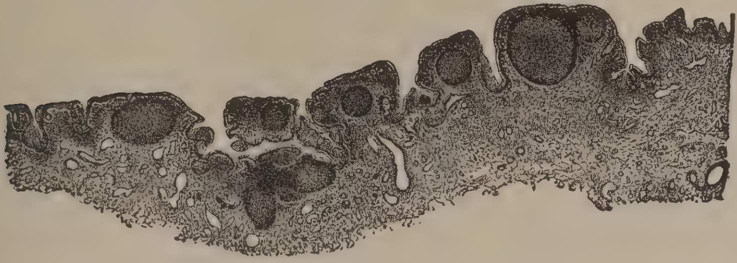


FIG. 19.—Trachoma

In the centre are cells, likewise lymphoid, but quite different. They are larger than the lymphocytes and their nuclei are not so strongly stained. They have been called epitheloid cells because they rather resemble in shape the epithelial cells. In fact they are leucocytes, the majority mononucleated, with a few polynucleated among them.

A third variety of cell that is regularly found in the trachoma granule should be given particular attention. A number of large cells, some stellate, some serrated or roundish, frequently furnished with long processes, are scattered about within the follicle. They contain all possible inclusions; remains of nuclei, brownish masses, residua of blood corpuscles, etc. The question of their importance has excited much discussion. The assertion of *Addario*, that they are hypertrophic, fixed connective tissue cells, seems to me to be unauthorized. They have been named corpuscle cells by *Leber*, because of their inclusions, and phagocytes by *Villard*. I can confirm the statement of *Junius* that these cells exhibit a sort of amœboid movement when examined in a living condition, taken from the fresh contents of a follicle, so they may be called phagocytic cells. These cells are not present in such large numbers in the follicles of an ordinary conjunctivitis follicularis. The follicles contain also red blood corpuscles, plasma cells and giant cells; all of them lying in the meshes of a fine connective tissue stroma.

The trachoma granule also contains blood and lymph vessels. The formation of a granule evidently begins with an accumulation of lymphoid cells in the meshes of the previously existing connective tissue stroma of the adenoid layer. Sometimes the connective tissue stroma becomes atrophic when the granules are very large, so it frequently appears to be very scanty.

Another debated question is whether the trachoma granule is enclosed in a connective tissue capsule, or not. A connective tissue membrane cannot always be made out in the earliest stages of the development of the follicle, but as it becomes larger the preexisting connective tissue fibres may be shoved aside and pressed together; whether, in addition to this, the fibres are increased in consequence of a reactive inflammation seems to be doubtful. I am unable to convince myself that such a thing takes place; on the contrary the periphery of the growing granule often appears to be relaxed. In most preparations of fresh trachoma the demarcation of the granule from the surrounding tissue is not sharply defined.

B. Changes in the Adenoid Layer of the Conjunctiva

A diffuse, great infiltration of small cells, chiefly mononucleated leucocytes, takes place throughout the entire conjunctiva simultaneously with a vascular hyperæmia. The whole of the conjunctiva tarsi is thickened, the papillæ are swollen, the conjunctiva of the fornix is much folded. Even the tarsus is involved, though opinions differ in regard to the manner in which the involvement takes place. There seems to be no question that the tarsus becomes infiltrated; tracts of infiltrating round cells, as well as discrete aggregations of the same, are to be found in it. It is also certain that the inflammation is much greater always in the subtarsal connective tissue, the adenoid layer, than in the tarsus itself. Even the subsequent contraction of the subtarsal tissue is much greater than that of the tarsus, which is slight in comparison. We must therefore suppose that the boat shaped curvature of the tarsus is in reality a result of the contraction of the subtarsal tissue. The idea has been advanced by *Straub* that the thick, cicatricial cord, frequently met with in the groove running parallel to the margin of the lid, represents the entire contracted tarsal conjunctiva, which has become retracted toward the margin of the lid. Cystic degeneration of the *Meibomian* glands is met with not infrequently in the tarsus.

Naturally it is very difficult to obtain an examination of the conjunctiva bulbi, because we try in every way to preserve it, but it has been proven to contain aggregations of lymphoid cells which resemble granules, especially near the transition folds.

The changes found in the epithelium are of less importance. They exhibit nothing specific, although we must suppose that the morbific agent of trachoma first settles upon, or perhaps more correctly, first enters through the epithelium. Most of the changes are those met with more or less in other inflammatory conditions of the conjunctiva. The epithelium may appear thickened by proliferation and in the cicatricial stage it assumes a character which resembles epidermis.

Sometimes there is a distinct increase of the goblet cells, which start in the deeper part of the epithelium, rise to the surface and empty their contents. In some places the epithelium can be seen to be necrotic. It may become overstretched by the subjacent granules and burst, so as to allow their contents to escape. A fact that has attracted the attention of all observers seems to me much more significant in the etiology of trachoma: the infiltration of the epithelial layer with wandering cells. This is well marked in nearly all sections, and it is plain that they loosen the normal texture of the epithelium and so favor the entrance of the specific trachoma agent into the adenoid layer.

Under the microscope the pannus of trachoma presents a vascular, very thick infiltration of lymphocytes, that extends forward from the margin of the cornea between the epithelium and *Bowman's* membrane. The latter may be destroyed and then the parenchyma becomes invaded. Neither the clinical, nor the anatomical examination throws any light on the way in which pannus is brought about. Some ophthalmologists believe it to be due to the irritation produced mechanically by the roughness of the upper lid, others ascribe it to a specific infection of trachoma. Both theories are plausible. We cannot expect to meet with follicles in the cornea, because the condition necessary for their origin, the adenoid layer, is absent. In old pannus we often see a thickening of the epithelium that resembles epidermis, or necrosis and ulcerations of the cornea.

The important question, whether we are able or not to diagnose trachoma by the pathological examination, is answered when we examine a slide prepared from the tissue of a benign follicular catarrh which recovers quickly under gentle treatment, without the formation of a scar. Follicles are present and an inflammatory infiltration of round cells occupies the adenoid layer, just as in trachoma, and we must admit that a positive differentiation between trachoma in an early stage and benign follicular conjunctivitis cannot be made in this manner. Yet I am convinced that the two diseases are separate and absolutely different. As we meet with the same kind of follicles in the simple catarrh produced by atropin, we are forced to the conclusion that the human conjunctiva reacts to the most various bacteriological and chemical irritations with the formation of the same inflammatory products, and is particularly fitted to do so through its adenoid layer. The inflammatory process in the conjunctiva is more marked in trachoma than in the other follicular diseases, but until the etiology has been cleared up we shall be obliged to define trachoma from the pathological, as well as from the clinical, standpoint as the follicular disease of the conjunctiva that results in cicatrization.

2. Sections from Old Cases of Trachoma

The several stages through which trachoma passes, with the advancing cicatrization and contraction of the conjunctiva, may be dealt with briefly.

Granules of various sizes are found. Their growth takes place in the periphery through the entrance of new lymphocytes from the lymph vessels. In the centre numerous caryocinetic figures may be seen, as here the cells multiply by indirect division. Another stage is represented by the gelatinous degeneration, in which the conjunctiva appears clinically gelatinized, or hyaline, and the granules seem to have disappeared in the thick swelling. The stroma of the granule becomes more distinct, while the cells begin to break down and empty spaces to appear. In this stage the epithelium also may show a sort of hyaline degeneration. In other cases a layer of tissue that has undergone hyaline degeneration is found beneath the epithelium. Meanwhile the infiltrating cells in the adenoid tissue become absorbed more and more, and are replaced by new connective tissue. Finally the follicles break down, and some may become partly absorbed without leaving cicatricial changes. Occasionally a follicle will burst and empty its contents into the conjunctival sac, but such spontaneous absorption without residua and such evacuation form rare exceptions and have not the importance in the cure and course of trachoma which has been ascribed to them by *Raehlmann.* The majority of the follicles undergo transformation into connective tissue. Fine bands of connective tissue appear and assume a peculiar, radiating arrangement about the granules. This tissue does not come from the lymphoid cells of the marginal zone of the granule, which are transformed into connective tissue fibres, as was formerly supposed, but is formed close to the vessels that surround the follicle. The cell elements of the granules die and are absorbed. Thus the granule first becomes enveloped in connective tissue, is crushed by it as it were, and at last connective tissue begins to form in its interior. The place of the follicle and of the formerly densely infiltrated adenoid layer of the conjunctiva is now occupied by a shrinking cicatricial tissue, in which the epithelium has lost to a great extent its cylindrical arrangement and is flattened like epidermis.

28. Social Aspect, Prophylaxis and Medicinal Treatment of Trachoma

If we would solve correctly the problems that confront us in the fight against, and in the treatment of trachoma we must briefly consider the social aspect of the disease.

I. Social Aspect of Trachoma

Trachoma is an infectious disease which seriously endangers the vision of many people. The trachoma patient suffers a considerable loss in his capacity for culture and business. Children with trachoma are unable to attend school satisfactorily, and are periodically excluded from attendance because of their inflamed eyes. The condition of an adult is much worse. When the disease has assumed a grave character his vision may be injured, or even lost, and his earning power very greatly impaired.

Trachoma is a serious concern for the community, for the family, the state and the nation. The disease once introduced into a family is accustomed to attack one member after another. The danger of infection is increased by crowded and unhygienic conditions. It is a disease of poor people. Add to this that trachoma begins so insidiously and gives rise to such slight symptoms, in very many cases, that the infected ones do not know for months, or even years, that they are diseased, and that in the meantime they are spreading infection. In trachoma mankind has to deal with a malicious fiend.

Trachoma is particularly grave when it affects persons liable to military duty, because it unfits them for army service and may endanger the defensive power of the people in many regions. When the trachoma patient finally becomes unable to follow his calling, as the result of his disease, provision for his support and care must be made by the state or community; this entails further economic trouble.

II. Systematic Fight Against Trachoma

These conditions, together with a statistical demonstration that the disease was spreading in the different provinces of the German empire, led to the adoption of a plan, which has the support of the government, for fighting trachoma. In accordance with this plan courses of instruction in the recognition and treatment of trachoma are given in many eye clinics and other places, especially in the eastern provinces, while inquiries are made systematically concerning its spread. Such an inquiry is not simple, for the entire population cannot be examined, but a general survey is obtained in three ways.

1. The ophthalmologists are able to detect any marked increase in the disease from the ratio of the number of patients with trachoma to the total number of patients with diseases of the eye.

2. The army surgeons report the number suffering from trachoma among the recruits, thus furnishing data concerning the degree of infection in the various districts.

3. A systematic examination of school children has proved the best gauge of the spread of the disease, and to this is added the examination of the people in different institutions, orphanages, factories, prisons, etc. The facts thus gathered prove the necessity that the fight against trachoma must be undertaken by the government, and efforts are constantly being made along this line.

The most important and efficient measure adopted is the regular control and treatment of the children in the infected schools by physicians appointed for the purpose. It is more difficult to treat regularly persons who do not attend school, even though they can be compelled to submit to treatment if they have trachoma. The more the people are enlightened concerning the importance of the disease, the better will be the prospect of its control. In regions where the infection is most prevalent every effort is made to call the attention of the populace to the danger, and it is the duty of every physician to teach that the transmission of the disease may be prevented by adequate cleanliness. In some places the teachers take part in the treatment of the children under the supervision of the physician.

The decrease of the disease in the Prussian army is an evidence that this systematic fight against trachoma may be successful. Trained nurses are employed in badly infected districts and the physicians have special hours of consultation for trachoma patients, while grave cases that require operative treatment are referred to the clinics and hospitals. But these measures alone are insufficient. We must protect ourselves against the continual importation of trachoma by foreign workmen, and there are laws that these foreigners must be examined. Yet, in spite of all, trachoma has not decreased among us. Elevation of the social position, improvement in the conditions of home life and, above all, education in cleanliness must be added to the systematic fight. But this is, and will long remain, a vain wish. Social misery is ever present and trachoma will not quickly die out.

III. Medicamental and Mechanical Treatment of Trachoma

As soon as the patient has entered the hospital he is provided with a wash basin and a towel for his sole personal use, and with two bowls, one containing a 1 : 5,000 solution of hydrargyrum oxycyanatum, with which he may bathe his eyes occasionally, the other holding a sublimate solution for the disinfection of his hands.

No remedy at our disposal is able to kill the hypothetical morbific agent without simultaneous injury to the tissues, and it remains for the future to discover a therapy based on the etiology. Under the guidance of *Paul Ehrlich* we are on the track of specific remedies which will sterilize, without injury to the tissues, organisms that have become infected with trypanosomata, and in this we may see the beginning of such a therapy. But as long as such an ideal therapia sterilisans is not at our disposal we must be content with other measures.

The large number of remedial measures that have been recommended may be divided into three general classes, the medicamental, the mechanical and the surgical forms of treatment. These may be combined one with another and in practice the medicamental and mechanical forms go together. The question whether an immediate operation is indicated or not arises with each patient. The condition of the eyes themselves, the form of the trachoma and the stage of the disease naturally are to be considered first in making the decision, but the social point of view must not be forgotten. In Greifswald the trachoma material consists almost wholly of Polish, or Russian, reapers, who are employed on the large estates. It is for the interest of the employer that the workman is not kept too long in the hospital and the receipt of letters requesting the discharge of these people is of daily occurrence. On the other hand the workman himself is in distress. He is earning nothing while he remains in the hospital, and he needs his earnings to support his family during the winter months. Hence his claim upon us is well founded that we should choose that mode of treatment which will effect a cure most quickly.

Another important point is that we must try not only to make him fit for work again as quickly as possible, but also to choose the mode of treatment best fitted, according to our clinical experience, to guard against relapses and reinfections. Trachoma tends to relapse, but the degree of this tendency varies with the different forms of treatment employed. One thing is certain: we can give the patient neither an absolute cure and freedom from the agent of trachoma, nor positive immunity. He is usually exposed to reinfection as soon as he returns to his infected dwelling and to association with trachomatous comrades.

Immediate surgical treatment is indicated 1, when a blepharospasm, a spasmodic contraction of the orbicularis, is present so as to render treatment of the trachomatous conjunctiva very difficult, whether the palpebral fissure is shortened or not; 2, when the lids are already in an abnormal position, practically when trichiasis and entropion are present; 3, when the lacrymal passages are diseased.

An obstinate blepharospasm must be relieved by a canthoplasty, otherwise it will delay the healing of the fundamental disease. Disease of the lacrymal passages demands surgical intervention for the same reason and as a rule nothing will serve except extirpation of the lacrymal sac. The presence of an entropion is even more strongly a positive indication that the treatment of the trachoma should be commenced with a surgical operation.

But when the first examination reveals no such condition in need of correction, the question again arises whether immediate surgical treatment of the conjunctiva is, or is not, indicated. The decision now depends on the stage of the trachoma and the condition of the diseased conjunctiva. One should become as proficient as possible in the most important methods of treating trachoma and should learn what each accomplishes, as well as to what forms and stages of the disease they are severally suited.

Medicamental Therapy of Trachoma

The first and most important object of medicamental treatment is to check the infectious secretion. Many patients seek relief simply from the abundant secretion, which may be viscid, mucous, or purulent, especially in the first stage of trachoma. The best means to control it is a 1 or 2% solution of silver nitrate applied to the conjunctiva and then neutralized with salt and water. After such an application the patient is given a bowl of cold boric acid solution, with which to cool and wash out his eyes as long as the irritation produced by the silver lasts. The eyes are then cleansed and irrigated by the nurse several times during the day.

During the evening sublimate vaseline (1:10,000) is applied to the margins of the lids and the neighboring skin, to prevent the sticking together of the lids in the morning and to protect the skin against erosions and soiling by the secretion. Patients should never be given the solution of silver nitrate to use at home because of the danger of argyrosis, which gives the eyes an extremely ugly appearance.

In many cases the increase of the secretion is due to mixed infection. In Germany trachoma is quite apt to be associated with the chronic blepharoconjunctivitis produced by diplobacilli, which may be recognized by its characteristic livid discoloration and inflammation of the canthus. In such cases frequent washings with weak solutions of zinc sulphate are indicated, and the sublimate ointment is replaced by a zinc ointment (zinc sulphate 0.04:10.0).

Trachoma patients exhibit very different degrees of tolerance toward silver nitrate. Sometimes the irritation produced is very severe; then a perfectly neutralized solution of lead acetate (1:100 or 1:200) may be employed. This should always be washed out again with water and must never be used when any corneal ulcer, or a commencing pannus, is present, because little, insoluble deposits of lead are formed in the tissue wherever there is a loss of substance, or a defect in the epithelium, and remain as permanent opacities.

Tannic acid, 0.1:10.0, may be used instead of silver in cases with a moderate degree of secretion, but I have seen no results from the use of the much praised protargol in trachoma.

After the secretion and the swelling of the conjunctiva have receded somewhat, the granules come into view more distinctly and the time has arrived for the mechanical treatment, which may be combined with the medicamental. The means at our disposal for this purpose are 1, massage of the conjunctiva; 2, the copper stick; 3, the alum stick.

1. Medicamental Massage in Trachoma

The procedure recommended by the *Keining* brothers is as follows: After the eye has been cocainized the lid is everted and the diseased conjunctiva is rubbed until it begins to bleed with a bit of absorbent cotton dipped in a 1:2,000 sublimate solution, or a 1:1,000 solution of hydrargyrum oxycyanatum. The eye is then bathed with boric acid solution and some cocain ointment is introduced to relieve the pain. Forceps wrapped with cotton may be used to enter the niches in the conjunctiva, which is an important part of the procedure. The harder the follicles, the harder must be the rubbing, but more care should be exercised when the conjunctiva is softened. This procedure is used extensively, not only in trachoma, but also in the treatment of harmless follicular diseases. The massage should be repeated as soon as the little superficial crusts which are produced have been thrown off. When carefully performed the massage may be repeated daily.

This massage is indicated from two points of view, first to produce a disinfectant effect upon the morbific agent, second to excite an absorption of the follicles, many of which are evacuated during the massage, by the irritation mechanically produced. It is self evident that superficial injuries of the conjunctiva result, but such are unavoidable by any means of mechanical therapy and are insufficient of themselves to condemn the procedure. We have only to consider whether the reaction excited by the mechanical irritation hastens the spontaneous cure of trachoma.

The advantages of this procedure are, that it is very simple and easily performed, that the contraction of the conjunctiva is unimportant, and that it can be used in ambulatory cases. But these are offset by serious disadvantages. The treatment has to be continued for a long time, in obstinate cases for months, and it affords no protection to the patient against a relapse, or a reinfection. *Kuhnt* has demonstrated this in many cases, and I believe he is right. Hence the use of massage is limited to mild cases that can come for treatment continuously, and to those in which its use follows operative intervention as an after treatment.

In another form of massage a ball provided with a glass stem is introduced beneath the lid, which is then massaged by counterpressure with the thumb, after sublimate vaseline has been applied. The transition folds may be rubbed back and forth horizontally with the little spatula of *Ottawa*, after the lid has been everted, and then, after the lid has been allowed to return to its normal position, the conjunctiva may be massaged. These methods, in my opinion, are less efficient than the massage with cotton and sublimate, but are useful to supplement other methods of treatment. The most useful application of massage is in the treatment of trachomatous opacities of the cornea.

2. The Copper Pencil

Next to massage the most important remedy of this class is cupric sulphate in the form of the copper pencil. Its surface must be perfectly smooth This is secured each time by rubbing the pencil on a moist cloth. Cupric sulphate is an astringent which promotes an absorption of the diffuse infiltration and of the follicles. It should be used when the secretion is gone, when the conjunctiva is no longer relaxed, as it is in the first stage, and when the papillary hypertrophy has become prominent. There is no doubt that this forms the best means to bring about an atrophy of the hypertrophic islands between the cicatricial bands. The presence of pannus forms no contraindication to its use, on the contrary, when the copper pencil does good service in the diseased conjunctiva an improvement frequently takes place in the pannus.

The application causes fairly severe pain, which may be controlled by cooling with boric acid solution and usually passes off in a short time, especially if the patient is in the open air. It may be used daily at first, then at longer intervals; patience is always needed. Cupric sulphate may be prescribed in the form of an ointment when it is necessary to give the patient a remedy for home treatment.

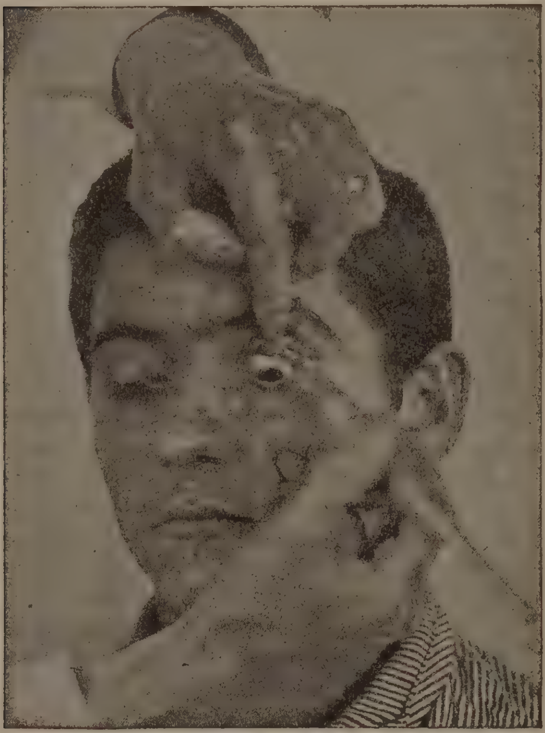


FIG. 20.—Application of the Alum Pencil.

3. The Alum Pencil

The alum pencil is of service as a substitute for the copper in the mechanical treatment of trachoma, in which it is well to change remedies frequently. Its smooth surface is applied to the diseased parts of the conjunctiva in the same manner; its action is milder, though it causes a sharp burning.

This completes the medicamental and mechanical therapy of trachoma most commonly in use at the present day. There remain only a few prescriptions for home use, for it is necessary to commit a portion of the treatment to the patients themselves, because of the long duration of the disease. The usual lotions prescribed are boric acid solution and solution of hydrargyrum oxycyanatum 1:5,000. The ordinary eye drops are zinc sulph., 0.04:10.0, borax 0.2:10, and copper glycerin (cupric sulphate 0.5, glycerin pur. 50.0, aqu. destill. 50.0). Ointments containing cupric acetate 5%, or orthoform 5 to 10%, may be given.

Medicamental therapy must be continuous for months, or even years, to obtain permanent results, because we have as yet no means by which to eradicate the agent of trachoma without injury to the tissues. The expert can protect himself against infection only by the most painstaking cleanliness, and when infection has once taken place it can be removed only in mild cases and by timely treatment. Because of this impotency great patience and energy are demanded from both the physician and the patient, and these are apt to be limited, so far as the patient is concerned, by the financial means. I do not share the optimism of those who anticipate the early prevention, or suppression of this disease by medicolegal measures, yet we must not falter. In the fight against trachoma we cannot spare the medicamental treatment.

29. The Operative Treatment of Trachoma

We meet with cases of trachoma in which we have no choice but to operate at once. Marked blepharospasm, entropion and disease of the lacrymal sac must be removed before the trachomatous conjunctiva can be treated either medicamentally, or surgically. Hence I shall deal first with measures to obviate these conditions. They include the enlargement of the palpebral fissure, the extirpation of the lacrymal sac and the operations for entropion and trichiasis.

Blepharospasm in Trachoma and Its Remedy

Very many trachomatous patients suffer from an obstinate blepharospasm, excited by the constant irritation of the inflamed conjunctiva, which causes severe subjective suffering, and at the same time favors the development and extension of pannus by the interference with the nutrition of the cornea produced by the increased pressure of the lid upon the anterior portion of the eyeball. In addition to this it aids in many cases the development of entropion. The spasmodic contraction of the orbicularis promotes the rounding off of the inner edge of the lid and a faulty position of the eyelashes, especially when the tarsus is diseased, softened and infiltrated, thus commencing a process which is finally completed as entropion by the cicatricial contraction of the conjunctiva.

Blepharospasm also facilitates the appearance of excoriations at the outer canthus, in consequence of the constant irritation produced by the increased secretion and lacrymal fluid. These in turn excite spastic contractions of the orbicularis and together they bring about a phimotic shortening of the palpebral fissure, which is accelerated by the contraction of the tarsus and conjunctiva. The blepharospasm must be corrected first, if we would make any progress therapeutically, whether the palpebral fissure is shortened or not. I no longer try to control it by instillations of cocain, or other medicamental means. The only way in which to obtain any real benefit is by an operative enlargement of the palpebral fissure. If a trachoma patient has dacryocystitis the lacrymal sac should be extirpated, likewise as a preliminary step. Entropion and trichiasis must also be corrected before other treatment is commenced. These operations will be dealt with in our studies concerning diseases of the lids.

The Operative Treatment of the Trachomatous Conjunctiva

The principal methods employed in the operative treatment of the trachomatous conjunctiva are 1, the galvanic cautery; 2, scarification and curetting of the conjunctiva; 3, expression of the granules; 4, excisions.

1. The Galvanic Cautery

This patient presents a mild case of trachoma in its initial stage. The cornea is intact, the granules are found only in the transition folds, lying not closely together, but distinctly separate.

The eye is cocainized and then, after the lid has been everted, 1 ccm of a 2% solution of cocain is injected beneath the upper transition fold, which immediately protrudes forward tensely, rendering the granules very distinct. The convex margin of the tarsus is seized with a pair of forceps, lifted up to the margin of the orbit, and now, when the patient looks downward, the entire transition fold and the smooth conjunctiva bulbi are in plain view, and a sharp line of demarcation can be seen between the thickened, granular transition fold and the healthy conjunctiva bulbi. Each granule that can be seen and reached is now cauterized with the platinum needle. The needle must be white hot, otherwise the effect will be insufficient and shreds of tissue may adhere to it, and it should be carried somewhat beneath the conjunctiva. In this way the visible granules in all four lids may be cauterized at a single sitting if necessary. After the operation the patient should lie down and an ointment of boric acid, or of cocain should be applied. The eschars are thrown off in a few days and the little funnel-shaped wounds cicatrize quickly.

This method of treatment possesses certain advantages. The subsequent irritation is slight, there is no danger of extensive cicatrization, and it can be employed in any stage of trachoma, in fresh cases with numerous follicles, or in old cases that have been treated in other ways, as long as individual granules can be found.

On the other hand it has serious disadvantages. A single treatment is not apt to suffice, but the cauterization must be repeated frequently. Of more importance is the clinical fact that malignant cases, especially those in which the cartilage is thickened and infiltrated, cannot be cured in this manner. Finally this operation furnishes no protection whatever against relapse and reinfection, a fact which has been established by *Kuhnt*. The galvanic cautery is fitted only for patients in whom the disease is mild, who can come regularly for treatment and are not compelled to return to a focus of infection.

2. Scarification aud Curetting of the Conjunctiva

In the next case another procedure will be followed.

Anæsthetization is produced in the same way by a subconjunctival injection of cocain. After the convex margin of the tarsus has been elevated the covering of the follicles is scarified with a small knife, or a scarificator, and the contents of each granule are removed by means of a little spoon. Some use for this purpose a metal pencil, or specially devised brushes.

This operation should be reserved for the small number of selected cases in which the conjunctiva surrounding the follicles is little altered. The conjunctiva should be given every possible protection during the removal of the contents of the follicles, but if these instruments, spoon, pencil, or brush, are used when the conjunctiva itself is thickened, infiltrated, fragile, or gelatinous, it will be seriously injured during the evacuation of the follicles, and cicatrization and contraction will be promoted. Hence it should never be employed except in mild cases, in which it may be productive of good results.

3. Knapp's Expression of the Granules

Two procedures that are much employed in the treatment of comparatively mild cases of trachoma are the expressions of the granules as performed by *Knapp* and by *Kuhnt*.

In the former, after thorough cocainization and eversion of the upper lid, one blade of *Knapp's* roller forceps is placed between the conjunctiva scleræ and the lid, the other on the everted surface of the latter. The blades are then pressed together and drawn several times over the same place. The soft, infiltrated masses of tissue are thus crushed between the rollers. Every part of the diseased conjunctiva that can be reached is thus treated, special attention being paid to the outer and inner corners. The conjunctival sac is then washed out, an ointment applied and the eye bandaged for some hours.

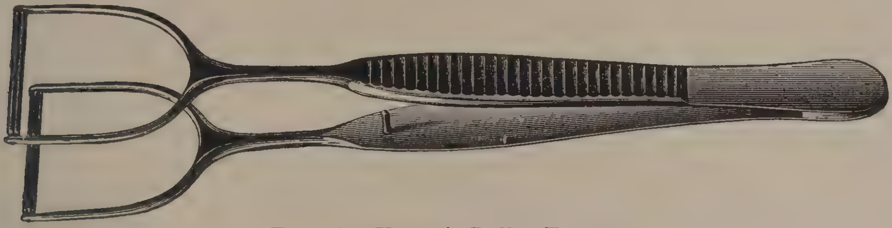


FIG. 21.—Knapp's Roller Forceps.

4. Kuhnt's Expression

In *Kuhnt's* operation a horn-plate is placed beneath the everted upper lid and a large number of punctures are made in the tarsus, particularly at its convex margin where the thickening is greatest. One blade of the expressor is then introduced into the transition fold and the other placed on the conjunctiva tarsi. The blades are then pressed together with considerable force, so as to cause the expression of gelatinous masses together with the contents of the granules.

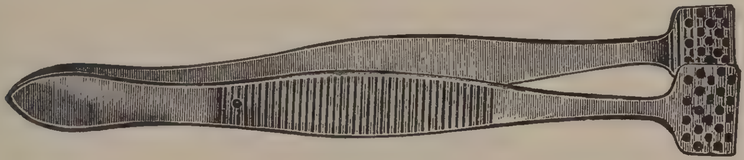


FIG. 22.—Kuhnt's Expressor.

These methods likewise have certain indications. Grave cases with gelatinous degeneration must not be treated in this manner, and the thickening of the tarsus must not be too far advanced. They must also be supplemented with medicamental treatment. They are particularly to be recommended in cases of native born patients who have become infected through foreign workmen, but live separately from them. In grave cases, and in the cases of foreign workmen who return to associate with infected comrades, excision is preferable. With these we enter the field of the specialist, for the procedures hitherto described may be practised very satisfactorily by the general practitioner.

5. The Excision Operations

The training of a specialist is necessary to learn the technique, and still more the indications for the three operations about to be described, but they should be known to every practitioner. Our trachoma material increases year by year, hence I am able to demonstrate these operations, in the technique of which I was instructed by my friend, *v. Blaskovicz* of Budapest.

The Simple Excision

When the trachoma has seriously invaded only the transition folds, and the patient has not sufficient time at his disposal for a mechanical, or medicamental treatment, simple excision of the transition folds may be performed. In these cases the convex margin of the tarsus must not be thickened or infiltrated.

After the subconjunctival injection of cocain the transition fold studded with follicles comes forward, and the sharp line of demarcation between the diseased area and the healthy conjunctiva scleræ can be plainly seen. The convex margin of the tarsus is seized with two pairs of forceps and an incision is made in the healthy conjunctiva scleræ, close to the line of demarcation, from the outer to the inner canthus. The wound immediately gapes and the conjunctiva scleræ retracts, so as to allow the bluish appearing *Mueller's* muscle to appear. Several sutures are then introduced carefully through the margin of the scleral conjunctiva, which is slightly undermined. A second incision is made separating the transition fold from the tarsus. The diseased transition fold is seized at the inner canthus, dissected free from the subjacent tissue with scissors and removed. The needles are then passed through the margin of the tarsus and the sutures tied. The conjunctival sac is washed out and the eye bandaged. If the margins of the wound have been accurately coapted no granulations form and only a slight scar is left. Improvement follows quickly when this operation has been performed on cases to which it is suited, and even a commencing pannus may pass away rapidly after the removal of the granular masses in the transition fold. But the operation accomplishes nothing if the tarsus is involved in the infiltration. In such cases a different procedure is required.

The Combined Excision

In this operation the diseased transition folds and a part of the infiltrated and thickened tarsus are removed together. The procedure is as follows:

The first incision is made as before, in healthy tissue close to the line of demarcation between the diseased transition fold and the healthy scleral conjunctiva; the wound gapes and the scleral conjunctiva retracts. Three sutures are then introduced at the margin of the scleral conjunctiva. The needle carrying the middle suture is passed from the wound outward through the conjunctiva close to its edge, and then passed back from without inward at a horizontal distance of 2 mm thus causing the loop of thread to lie on the epithelial surface of the conjunctiva. The other sutures are inserted in the same way in the outer and inner thirds of the margin of the wound. These sutures are now held firmly in the hand and traction made upon them while the convex margin of the tarsus is drawn upward and the scleral conjunctiva is dissected far down with scissors. Now comes the second step of the operation. *Jæger's* plate is placed beneath the skin surface of the singly everted lid, the margin of which is pressed firmly upon it with

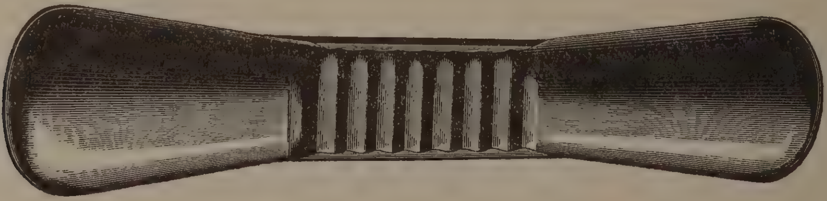


FIG. 23.—Jæger's Lid Plate.

the first two fingers of the left hand, or the margin of the lid may be seized with *Bloemer's* forceps and the tarsal portion of the lid kept tightly stretched. How much of the diseased tarsus we should remove depends on the nature of the case, the severity and extent of the trachoma. The incision through the conjunctiva and tarsus joins the inner and outer ends of the first incision by an almost straight line running parallel to and 2.5 to 3 mm from the margin of the lid, and delimits the entire piece of diseased conjunctiva and cartilage. This must next be dissected up without injury to the orbicularis, or to *Mueller's* muscle. The inner end of the tarsus is lifted up with forceps and dissected free from the orbicularis, then the separation of the conjunctiva in the transition fold is completed and the tarsus is parted from the insertion of the smooth muscles. The piece removed measures about 2.5 cm long by about 1 cm broad. Everything depends now on the exact coaptation of the loosened conjunctiva scleræ with the cut edge of the cartilage in order to close the wound.

The middle suture is drawn downward and forward, after the patient has opened and closed the eye a few times. The margin of the lid is seized with forceps at the place where the suture appears beneath it, is lifted up somewhat, the point of the needle is slid along the forceps over the conjunctiva of the stump of the tarsus until it glides over the cut edge of the tarsus, and is then passed 1 mm higher through the lid from within outward. The other sutures are similarly inserted in a radiating direction. The margin of the lid is lifted up again, that we may satisfy ourselves that no folds have been formed in the conjunctiva, and then the sutures are tied quite loosely. The sutures may be removed at the end of three or four days.

This operation also should be performed only on selected cases. It is indicated in those forms of trachoma which exhibit a marked infiltration of the tarsus in addition to the development of granules and hypertrophy of the mucous membrane, particularly when the cornea is involved. The operation may be advisable even when the cornea is still intact, if it seems to be threatened by an extensive disease of the transition folds. The conjunctiva scleræ must be healthy and show a sharp line of demarcation from the diseased tissue.

Many objections have been made to such a sacrifice of the conjunctiva and a large part of the tarsus. One that has been raised is that "by it tissue is sacrificed which would not be completely destroyed by the disease" (*Sæmisch*). It has been urged that symblepharon has resulted in not a few cases. Recurrences have also been observed to take place after it had been performed. But clinical experience has proved that the proper use of this operation marks a great therapeutic advance. One must have seen how beneficially these operations can act in order to become an advocate. In grave cases of trachoma the tarsus gradually undergoes a degeneration and contraction which in turn inflicts serious injury upon the eye, and the principal focus of the infection is removed with the transition fold. On both of these grounds the objections to the operation are weak. Then consider that the course of the disease is shortened very much in the majority of cases; that after the excision pannus clears up very rapidly; that the development of entropion is prevented; and finally that observations indicate that relapse is guarded against in a large proportion of the patients operated on. It may be said that about 50% of the grave cases of trachoma may be improved or cured by this operative measure.

Like most operations this one has its disadvantages. Complications may occur. Above all it is necessary to keep rigorously within the indications for the operation of excision, to reserve it for grave cases, and to perform it with the utmost exactitude.

Enucleation of the Tarsus

Kuhnt's operation of enucleation of the tarsus is of service in cases of cicatricial trachoma with advanced degeneration and abnormal curvature of the tarsus. After eversion a plate is placed beneath the skin surface of the lid, the margin of which is drawn firmly upward. An incision is made parallel with and close to the margin of the lid through the conjunctiva and tarsus. The conjunctiva recedes somewhat, and the lower surface of the tarsus is separated from the connective tissue as far as the convex margin. The tarsus is then seized and separated from the conjunctiva, but usually a small strip of the latter remains attached. When this dissection has been carried as high as the upper border of the tarsus the insertion of the levator palpebræ is divided, beginning at the inner canthus, and the excised portion is removed. The conjunctiva is then sutured to the cut margin of the tarsus. Not only is blepharoptosis lessened, but entropion and inversion of the lashes is corrected and *v. Blaskovicz* considers that the greatest advantage afforded by this operation is its effect on trichiasis. The operation is indicated when the conjunctiva is wholly, or in great part cicatricial and the tarsus is so thickened or distorted as to produce an inversion of the cilia, an entropion with trichiasis.

The short time at our disposal for the consideration of trachoma is insufficient for exhaustive instruction in its treatment, or for more than a general survey of the subject. Trachoma varies in malignancy, form and duration in different regions. Its character and curability vary to an extraordinary degree. One therapy does not do for all cases. Choice must be made from the various measures in the armamentarium by every one who practices in a trachomatous region. The subject may be closed with the golden words of *Kuhnt*, our best authority on trachoma: "The chief precept of therapeusis demands careful individualization. The physician should treat no concept of disease schematically, but organs in a certain stage of disease with regard to their natural relations with other organs, with the entire sick man, and the dependence of the latter on external conditions. If this truth should be borne in mind anywhere, it should be in the treatment of trachoma."

30. The Benign Follicular Catarrhs of the Conjunctiva

We have considered in detail the development and importance of the follicle in connection with trachoma, but, as we meet with follicles in harmless diseases of the eye, it is desirable for diagnostic reasons that these harmless follicular diseases should be briefly considered.

Benign, or harmless follicular diseases of the conjunctiva are those in which follicles form as in trachoma, but disappear without having caused cicatrization. This differentiation must be firmly fixed in mind.

The difference between trachoma and benign conjunctivitis follicularis is the more remarkable inasmuch as the follicles have the same anatomical structure in each disease. The follicles are situated in the adenoid layer of the conjunctiva and consist of lymphoid cells, lymphocytes and leucocytes, which lie discretely embedded in a fine connective tissue stroma without a firm capsule. In both diseases they elevate somewhat the epithelial layer.

Yet the course and termination of the two diseases are so different that we must consider them essentially unlike. It is true that the diagnosis cannot be made with certainty in all cases as long as the agent of trachoma cannot be recognized, and that frequently the differentiation can be made only by the course and the results of treatment. The conclusion that the mucous membrane reacts to various influences with the formation of the same product may be drawn from the fact that the same follicles appear in two such different diseases. The specific influence of the disease itself finally settles the termination in each case either in cicatrization and destruction, or in restitutio ad integrum.

I

There are several groups of conjunctival troubles in which such follicles are formed.

Group 1; Follicles in Acute Catarrhal Conjunctivitis

Follicles may appear in the conjunctiva during the course of many acute infectious inflammations of the conjunctiva, such as a pneumococcal inflammation, or the conjunctivitis caused by the *Koch-Weeks* bacillus, usually after the abatement of the acute inflammatory symptoms. They appear as a number of smooth, whitish red prominences varying in size from 1 to 3 mm, usually situated in the lower transition fold, where, if few in number, they are gathered together about the canthi. They are to be found more rarely in the upper transition fold, but it is incorrect to maintain that they appear only in the lower transition fold in benign folliculosis. Their formation may extend from the lower transition fold into the plica semilunaris. Swelling and hypertrophy of the papillæ are present in addition to the follicles. Folliculosis can also be met with in the course of simple chronic catarrhal conjunctivitis and in other forms of inflammation that owe their origin to the most widely different agencies.

Group 2; Harmless Folliculosis in School Children

In many school children the conjunctiva bulbi is very slightly reddened, the vessels fairly distinct only in the periphery toward the transition folds, the conjunctiva tarsi is moderately red, not thickened, and allows the *Meibomian* glands to shine through distinctly. In the lower transition fold lie little rows of follicles, particularly dense toward the canthi. The eyes are absolutely unirritated; the majority of the children are unaware from their sensations that they have any pathological changes in their eyes. This absence of symptoms is directly disproportionate to the marked development of the follicles.

Greeff and *Meyweg* have shown that these follicular diseases are not communicable. Yet these ''school follicles'' are very often mistaken for trachoma. This form of folliculosis is met with very frequently; it spreads almost like an epidemic in schools and among the inmates of orphan asylums, barracks, etc.

The origin of this follicle is evidently to be ascribed to chemico-physical injuries. School children remain for hours in rooms the air of which is not unobjectionable, to which is to be added overexertion and a special predisposition among young people. Wherever numerous individuals are crowded together, as in schools, a moderate amount of this folliculosis may be expected. School physicians should be careful with their rigorous measures, such as closing the schools, when dealing with this condition.

The question has arisen, in view of their frequent occurrence, whether follicles do not preexist in the normal conjunctiva. This has not yet been definitely determined. Some authors recognize physiologically embryological rudiments, others have been able to find no lymph follicles in the normal conjunctiva.

Some ophthalmologists think that other benign chronic folliculoses exist which are infectious. The condition is bacteriologically negative and it must be supposed that in addition to the agent of trachoma, which excites its specific disease, there are other germs, belonging perhaps to the same group, which excite these benign and infectious follicular diseases. *Axenfeld* obtained on his own conjunctiva a slight folliculosis that healed spontaneously, through inoculation in an endemic in an orphan asylum. The question concerning such agents of endemic, benign follicular diseases, so far as they do not appear as annexed to known infections, is still open.

A third form of harmless folliculosis sometimes appears in patients who have been treated with atropin for weeks and have borne the drug well. It starts with a sharp conjunctivitis in which no bacterial cause can be detected. The lids are somewhat swollen, there is a moderate dermatitis at the inner canthus and the lacrymal secretion is increased. Rows of follicles lie in the lower transition fold. The conjunctiva itself has a peculiar rose red, or yellowish appearance. The prolonged use of atropin has finally excited in the conjunctiva a reaction associated with folliculosis and inflammation of the skin of the lid. It is called atropin catarrh, but the same folliculosis may be excited by the use of eserin, or of unguentum hydrargyri oxidi rubri. When the remedy is used in only one eye, the folliculosis is confined to that eye. These examples show the idiosyncrasy of the conjunctiva to chemical poisons.

II

How can these harmless folliculoses be distinguished from trachoma? A positive differentiation cannot be made at an early stage until we are able to demonstrate the agent of trachoma with certainty and ease. Until that time arrives many of these cases will need to be designated "trachoma (?)" and we shall have to wait for the course of the disease to clear up the diagnosis.

Recovery is delayed even in the harmless follicular diseases by the presence of the follicles, but yet takes place much more quickly than in trachoma. Two symptoms are characteristic of the benign follicular diseases: The follicles undergo complete involution and leave not a trace of cicatricial tissue, and the deeper tissues of the lids, the tarsus, and the cornea remain perfectly free from the diseases we are accustomed to meet with in them in trachoma. The prognosis of conjunctivitis follicularis is good.

III. Treatment of Benign Follicular Diseases

In atropin catarrh the use of the drug is discontinued; follicles that appear in the course of an acute catarrh disappear under the ordinary treatment. In the other cases, particularly those of school children, bathing the eyes with boric acid solution will suffice, but in obstinate cases we may use 1, instillations of hydrarg. oxycyanat. 1:2,000; 2, the alum pencil, with which the follicles are wiped two or three times a week; 3, gentle massage with cotton wet with sublimate 1:1,000 applied in the same way as in trachoma.

31. The Clinical Picture of Keratoconjunctivitis Eczematosa and its Connection with Scrofulosis

Sometimes the opinion is frankly expressed that it is really unnecessary for the general practitioner to study ophthalmology. The public itself demands the treatment of a specialist in diseases of the eye and there is not much left, therapeutically at least, for him. Therefore, without special training, he can be of the best service when he sends all eye cases as quickly as possible to the specialist. There is some truth in these criticisms, for, with the increasing development of special lines and the growing differentiation of medical work, the demand of the public for treatment by specialists is steadily growing louder. The limits to which the general practitioner is confined, in the therapeusis of the eye, are narrow. Yet it is necessary that he should be familiar with ophthalmology. He needs to be able to recognize the most important diseases of the eye, and he can accomplish much good, even therapeutically, when he has learned to treat certain diseases of the eye independently. One of the most important, as well as most frequent, of these diseases is keratoconjunctivitis eczematosa, which is to be seen in some of its manifold manifestations almost daily in most clinics.

This is a specific disease of the surface of the eye which affects both the cornea and the conjunctiva, as its name implies, and very frequently appears at the place where the conjunctiva joins the surface of the cornea. Sometimes the cornea alone is involved. It differs fundamentally from the other diffuse inflammations of the conjunctiva, in that it is discrete in character and never involves more than a portion of the surface of the eye. Hence conjunctivitis eczematosa in its pure form is not accompanied by an abnormal secretion, like that met with in diffuse inflammations. Just as the follicle and the process of contraction are clinically diagnostic in trachoma, so the nodules form clinically the fundamental characteristic of keratoconjunctivitis eczematosa, but the nodules differ in appearance as they are situated on the conjunctiva, or on the cornea. They should therefore be studied carefully, and we will begin with the efflorescences that appertain to the conjunctiva.

I. Study of the Efflorescences in the Conjunctiva Bulbi

When the bulbar conjunctiva is the seat of the disease the nodules may appear either at the margin of the cornea, exactly in the limbus, or more peripherally in the conjunctiva bulbi proper.

Cases from Group A.

The simplest type of conjunctivitis eczematosa is the solitary efflorescence at the limbus. This child's eye is normal except for a roundish, or oval, whitish gray nodule about as large as the head of a pin at the margin of the cornea, which is distinctly elevated above the surrounding surface. Some hyperæmic vessels of the conjunctiva form a triangle with the nodule at its apex. The rest of the conjunctiva is perfectly well, and the discrete character of the disease is plainly evident.

Even in the most modern textbooks, this formation is called a phlyctæna, and the disease conjunctivitis phlyctænulosa, names bestowed many years ago, but which are erroneous; phlyctæna means vesicle, while this efflorescence is not a vesicle, but a subepithelial nodule. It consists of a heap of round cells beneath the elevated epithelium, with which it is in close contact. Toward the centre the cells often increase in size so much that they may even resemble giant cells.

This nodule shoots up very rapidly at the limbus, presses up the epithelium and attains its growth in a few days. Then its upper surface softens, the epithelium is partially thrown off and a little ulcer with a whitish gray base is formed, still above the level of the conjunctiva. These ulcers usually get well very quickly, are covered by fresh epithelium, the infiltration is absorbed, the nodule disappears completely and the injection of the conjunctival vessels passes away. No trace remains at the end of from eight to fourteen days, for these efflorescences heal without permanent damage to the eye as long as they are not situated on the cornea.

Manifold variations of this, the simplest type of conjunctivitis eczematosa are met with. In the first place the *number* varies. A patient may exhibit two nodules at the margin of the cornea, each with its own injection of the conjunctiva, or three, when the injections run together somewhat, or the number may be very great, when the injection of the conjunctiva is reticulated and diffuse, so that the entire eyeball looks red. The more numerous the nodules the smaller they are; this leads to the fact that they vary not only in number but also in *size*.

Another patient has a single efflorescence at the margin of the cornea which is nearly as large as a lentil. Such nodes are designated as broad marginal efflorescences. They are more obstinate in their course, require a longer time for ulceration and absorption, and are accompanied by a much greater hyperæmia. Such nodes are met with rarely.

The picture in the next patient forms a striking contrast; the efflorescences are extremely small, so that they give the entire limbus a granular appearance, and the conjunctival injection is very great. In such cases the nodules are very numerous, and it may be noted again that the smaller they are the greater is their number. They may be so small that they can scarcely be perceived with the naked eye while the entire limbus seems to be raised. We call this form of eczematous conjunctivitis "sand grain (sandkornförmige) efflorescences."

Cases from Group B.

The nodules in keratoconjunctivitis eczematosa vary not only in their number and size, but also in their situation. They may appear at a distance from the limbus in either the conjunctiva, or the cornea. Here is a case in which a large efflorescence of this nature is to be seen in the conjunctiva at a distance from the limbus, surrounded by a marked injection. The stage of an eczematous efflorescence should be recognized at a glance. In this case the ulceration of the surface is easily to be seen. Such a case, in which the elevation of the conjunctiva bulbi is superficial, was formerly known as an ulcus elevatum. The efflorescences may appear in all parts of the bulbar conjunctiva, but usually they are either near the margin of the cornea, or in the zone of the insertions of the muscles.

In very rare cases true efflorescences may appear in the tarsal conjunctiva near the margin of the lid; they are known as efflorescences of the lid.

II. Keratitis Eczematosa

When the cornea is involved four distinct clinical pictures are produced which may be present in any given case either singly, or in various combinations. These fundamental types are 1, the eczematous infiltration; 2, the eczematous ulcus rotundum corneæ; 3, the eczematous ulcus migrans; 4, the eczematous pannus.

Our next patient has a little, roundish, superficial opacity in the cornea. It has no vessels and is covered by epithelium, perhaps slightly elevated; evidently an infiltrate. The cornea is surrounded by an injection. This infiltrate is a nodule. It consists of an aggregation of cells beneath *Descemet's* membrane. It may undergo absorption, leaving little or no opacity in the cornea, or it may break through *Bowman's* membrane and produce an ulcer.

These eczematous nodules may appear singly, or in great numbers. When they occur in the marginal portion of the cornea the case is said to be one of marginal keratitis. They are very common, both in this situation and in the central part.

Like the conjunctival efflorescences they differ much in size. Sometimes the surface of the cornea may be studded with very small ones, which cause minute losses of substance after the epithelial covering has broken down. These are the same as the sandgrain efflorescences met with in the limbus, and, in the initial stage, the cornea looks as if it were sprinkled with sand. Thus it is evident that the fundamenal characteristic of conjunctivitis eczematosa, the inflammatory nodule, is likewise met with in the cornea.

The examination of efflorescences in the conjunctiva alone is easy, but kerato-conjunctivitis eczematosa is apt to excite marked photophobia and blepharospasm, because the terminal filaments of the nerves in the cornea are lacerated when the epithelium is lifted up by the eczematous eruption, are irritated also by the inflammation, and cause the blepharospasm through reflex action. The thought of conjunctivitis eczematosa is suggested at once when a child is seen to be suffering from photophobia.

In the next child we find near the margin of the cornea a roundish, cuplike ulcer, which extends deeply into the parenchyma and has its margins densely infiltrated. This is the ulcus eczematosum. It has characteristic features, is roundish, may appear in any part of the cornea, and represents an advanced stage of the eczematous nodular infiltrate, in which the process is more malignant. The infiltration soon becomes purulent, assumes a yellowish, almost pustular appearance, rapidly breaks down and produces ulceration. It hardly needs to be mentioned that iritis coexists.

This form of ulcer has a characteristic tendency to perforate the cornea. Although it may appear anywhere in that tissue, it shows a certain preference for the marginal portion, in front of the iris. When such an ulcer perforates, the iris falls forward and forms a prolapse. Healing takes place with the formation of a leucoma adhærens, which remains throughout life. I call this ulcer ulcus eczematosum rotundum because of its shape. Often it is met with in one eye alone.

Aside from its tendency to perforate, the eczematous corneal ulcer is of serious importance on account of the cicatrix it leaves. All of the maculæ corneæ seen in these children are the scars left after the healing of such ulcers. In adults they form one of the most frequent causes of poor vision.

The identification of the conjunctival with the corneal efflorescence has been rendered difficult, in our study thus far of the fundamental characteristics of these eczematous diseases, by one circumstance: a characteristic injection of the bloodvessels is associated with the efflorescences in the conjunctiva, while no such vascularization is formed in the cornea with either the nodular infiltration, or the fresh ulcus rotundum. But here is a case with an oval, or roundish, eczematous ulcer at the margin of the cornea to which superficial vessels extend from the thick, red, puffy looking limbus. *It is a characteristic peculiarity of the eczematous corneal diseases that they tend to become vascularized from the conjunctiva bulbi.* Thus it happens that vascularization is frequently met with in all possible forms of eczematous keratitis.

In this child's cornea is a narrow, tonguelike opacity, so characteristic that it cannot be mistaken for anything else. In it run two little bloodvessels and a crescentic nodule lies at its apex, near the centre of the cornea. This has been called keratitis fascicularis, or fasciculosa. In such cases a nodular infiltration is formed primarily at the margin of the cornea, and breaks down into an ulcer to which vessels extend from the margin. The peripheral portion heals, but on the opposite side the ulcer spreads toward the centre of the cornea, pushing before it its infiltrated edge, the gray crescent. As the infiltration and subsequent breaking down extends, the vessels follow and a narrow groove is formed which is filled by them. I call this form of eczematous corneal disease ulcus eczematosa migrans.

The course is slow, often lasting for weeks, while the ulcer may extend to, and even beyond the centre of the cornea. It differs from other eczematous ulcers in that it always remains superficial and never perforates, but it leaves a permanent, narrow, cicatricial band of opacity. Sometimes this opacity is forked, showing that the ulceration with the vessels advanced in two divergent directions.

In the last form of keratitis eczematosa nearly the whole of the cornea is reddened by a superficial vascularization with its typical ramification. This is pannus eczematosus. Numerous superficial infiltrations appear in the cornea, soon become confluent and give rise to the great vascularization. Between the vascular twigs the cornea can be seen to be not clear.

These nine different types of keratoconjunctivitis eczematosa are not always met with in such distinctive forms, more often they are combined together in various ways. Thus we find marginal efflorescences with old corneal maculæ, infiltrates and ulcers in one cornea, pannus and conjunctival efflorescences in another, and so on. The clinical picture of the disease is extremely variable.

III. The Habitus Scrofulosus and its Relations to Keratoconjunctivitis Eczematosa

No adequate conception of the entire clinical picture of this disease can ever be obtained from a study of the changes on the eyeball alone; many things are striking when you look at the patient placed before you.

In the first place keratoconjunctivitis eczematosa is a disease of childhood which appears latest at the time of puberty, although persons who have had it in childhood may have to deal with it later.

Another important feature is its great tendency to relapse. The efflorescences in the conjunctiva disappear in a short time, while the troubles in the cornea last longer, but in very many cases one attack follows another, so that the disease persists for years, often continuing until the time of puberty, when the attacks become less frequent and finally cease.

Two symptoms are more or less marked during the fresh attacks in nearly all such cases: photophobia and blepharospasm. These produce by reflex action an increased lacrymal secretion, which may be considered a third symptom.

The constant overflow of tears irritates the margins of the lids and frequently excites a blepharitis, while the skin becomes sore, and little excoriations appear at the outer canthus which are extremely painful and maintain in turn the blepharospasm. These must be taken into account in every case.

In some cases the skin of the lids and face is eczematous, the eczema often having an impetiginous character. Eczema is particularly apt to be found on the scalp. The thickness of the lips and nose is often striking. *All these things indicate that keratoconjunctivitis eczematosa is related to the so called scrofulous diathesis.* The nature of scrofulosis, particularly its connection with tuberculosis, has been much discussed. With many other clinicians I believe that scrofulosis is a disease sui generis, which is complicated secondarily with tuberculosis in many cases, and agree with *Grawitz* that it is a "disturbance of nutrition with a special tendency to inflammation of the lymphatic system." It is not my duty in an ophthalmological clinic to present an exhaustive survey of scrofulosis, but we will consider briefly the symptoms of the disease.

Scrofulosis appears under two forms, the torpid and the erethistic types. In the torpid, or sluggish, type the children appear coarse and bloated, with pale skins, thick noses, puffy lips, short, thick necks and numerous enlarged lymphatic glands. They are slow, both physically and mentally. In the erethistic, or nervous, type the children are of slight build, are lacking in subcutaneous fat, and usually have good mental capacity, but their skins are pale, reddening and paling very readily, and their lymphatic glands are distinctly swollen.

The lymphatic glands of the submaxillary, jugular and auricular regions in particular become swollen at an early period. In small children the retropharyngeal lymphatic glands deserve notice, because retropharyngeal abscesses may originate in them.

Czerny has called attention to the glandulæ occipitales in connection with catarrhs of the upper air passages. The bronchial glands may become greatly enlarged and give rise to a desire to cough; sometimes a dilation of the pupil on the affected side is produced by the pressure of these enlarged glands upon the sympathetic nerve.

One of the most important symptoms associated with the swellings of the lymphatic glands is the recurrent nasal catarrh, the secretion from which irritates the neighboring skin and causes scrofulous eczema to appear on the nose and upper lip. On the latter the inflammation may involve the subcutaneous tissue, render the lip thick and favor the formation of fissures. The eczematous places in the skin form favorable soils for the development of impetiginous diseases.

Besides the chronic rhinitis, scrofulous children have inflammation of the lymphatics in the throat; hyperplasia of the faucial tonsils and chronic pharyngitis. In addition to this the inflammation may extend up the Eustachian tube and cause catarrh of the middle ear.

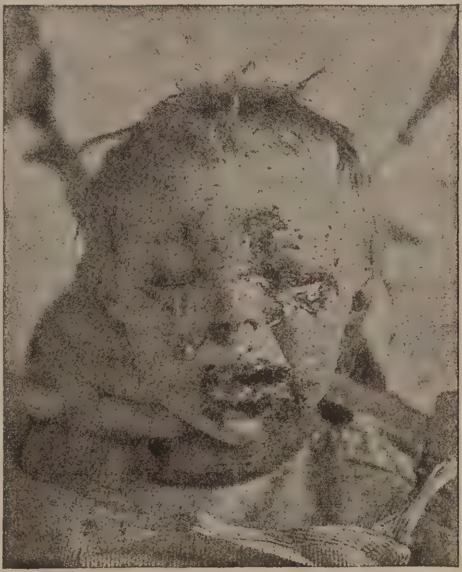


FIG. 24.—Keratitis Eczematosa with Scrofulosis.

Leucoplakia on the tongue, an abnormal, uneven abrasion of the epithelium, is a frequent symptom of scrofulosis. In both forms the functional activity of the sebaceous and sudoriparous glands of the skin is increased. This explains the susceptibility of the skin to eczema, which attacks by preference the scalp and the skin of the face, but in regard to this we must often take into account the part played by pediculi.

It should also be mentioned that the mucous membranes of the respiratory and intestinal tracts suffer in various ways; the digestive troubles especially may be very varied. Lastly, of the general symptoms we should not forget the secondary anæmia (diminution of the red blood corpuscles) and the leucocytosis which result from the existing suppurations. Even as ophthalmologists we have to study carefully the general condition of our patients, but the changes in the eyes typical of scrofulosis are those which preeminently claim our attention to-day. Finally I place before you a child in which the relations of keratoconjunctivitis eczematosa to the habitus scrofulosus can be pointed out.

This anæmic, ill nourished, but bloated-looking child has a very marked blepharospasm. The lids are thick, the veins in the lid skin are quite tortuous and bluish, while the skin is excoriated at the outer canthus. We call these excoriations rhagades. They are one of the most frequent causes of photophobia and blepharospasm. On the head is an extensive, moist eczema; in many cases pediculosis is the primary cause of this. The ears are eczematous and covered with scabs. The nose and lips are swollen, the mouth has a snoutlike appearance and the nares are eczematous and fissured. Changes which may exist within the nose and the nasopharyngeal space must be taken into consideration in our therapeusis. The glands about the lower jaw, in the neck and in front of the ear are swollen.

If we leave the child to himself he creeps into a dark corner of the room. In bed he turns away from the light and buries his face in the pillow. At the present time he covers his face with his hands and struggles to prevent the examination of his eyes, because he suffers pain every time they are opened, as long as the rhagades persist at the canthus. This picture exemplifies the relations of scrofulosis to keratoconjunctivitis eczematosa. The intensity of the photophobia is often disproportional to the severity of the disease. It is often slight with grave corneal ulcers, while it may be very great with simple efflorescences and infiltrations. Occasionally the blepharospasm is so great that the upper lid becomes everted, in consequence of the constant muscular contraction, and then the exposed mucous membrane becomes enormously swollen through the stasis of its circulation.

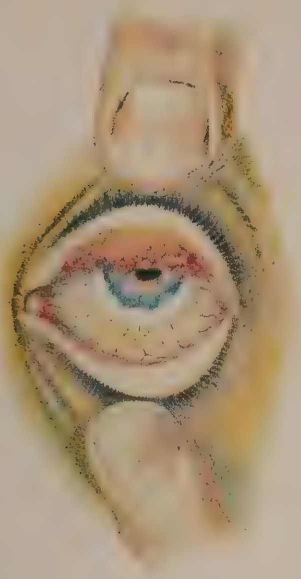
Although the lesions are fortunately not so serious in all cases, as in this last one, yet keratoconjunctivitis eczematosa may be easily recognized as it is the commonest disease of the eye. It is present in from 20 to 40%, or even more, of the total number of patients. It rarely attacks an apparently healthy child. The great majority of patients exhibit more or less distinctly the characteristic symptoms of scrofulosis, but sometimes they are slightly marked.

32. Cause, Course and Treatment of Keratoconjunctivitis Eczematosa

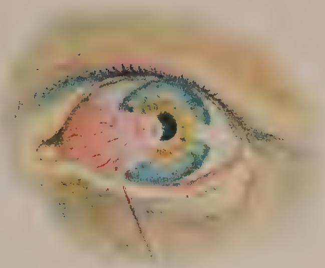
We have now studied the clinical picture of keratoconjunctivitis eczematosa as it appears in the conjunctiva and cornea and have found that it has a certain connection with scrofulosis. We have still to discuss this connection, the course of the disease and the treatment. The latter is most necessary. It has never been my experience that the medical student was able to attain the same perfection, in this respect, from the summary presentation of the subject in textbooks, or from the theoretical enumeration of the methods of treatment in the clinic, as that possessed by the general practitioner.

I. What is the Cause of Conjunctivitis Eczematosa?

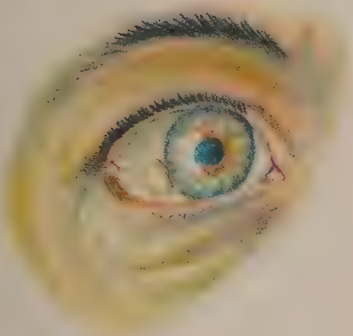
Although a certain connection exists, scrofulosis is not necessarily the only cause of keratoconjunctivitis eczematosa. All scrofulous children do not suffer from this disease and, on the other hand, we meet with cases in which positive signs of scrofulosis cannot be demonstrated, even though it cannot be excluded. But it is distinctly characteristic of the disease to attack such children intermittently during the years that they are subject to scrofulosis. All that we can say as yet is that in the majority of cases the scrofulous diathesis predisposes to this disease. Concerning its etiology the question has arisen whether the subepithelial nodules



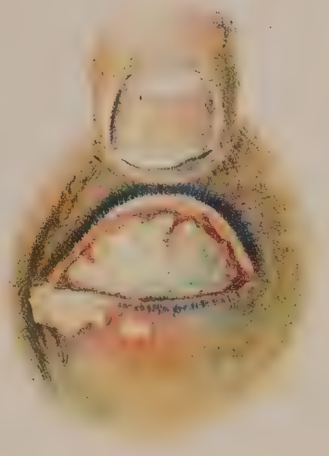
a Spring catarrh.



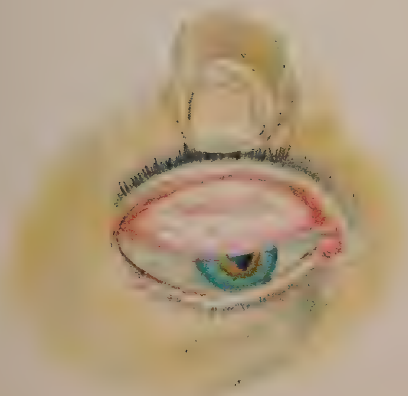
b Pterygium, Sound passing under its neck.



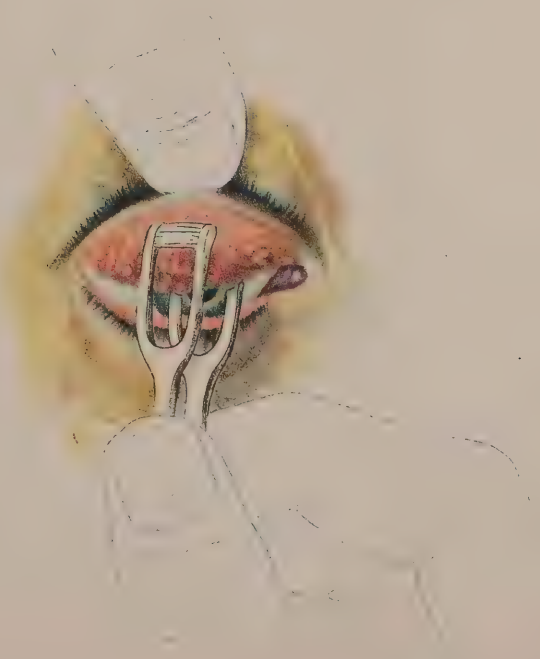
c Dermoid of the conjunctiva.



d Conjunctivitis crouposa.



e Cicatricial trachoma.



f Expression of trachoma granules.

Rebman Company, New York.

of infiltration in the conjunctiva and cornea, which form its characteristic feature, originate through ectogenous, or endogenous infection.

The ophthalmologists who believe the origin to be endogenous point out that endogenous forms of conjunctivitis exist, such as the gonorrhœal conjunctivitis produced by metastasis from urethral gonorrhœa. *E. Cohn* produced experimentally a conjunctivitis with decomposed yeast. But such conjunctival diseases as these cannot be compared offhand with the discrete efflorescences just beneath the epithelium. The fact that circumscribed lesions are formed in the transparent cornea by herpes zoster and herpes febrilis is not pertinent to the question. The experimental attempts to excite the disease endogenously must be considered failures. *Leber* and *Bruns* injected dead tubercle bacilli into the veins of rabbits and then examined the eyes for metastatic lesions; but the results they obtained proved neither the endogenous origin of keratoconjunctivitis eczematosa, nor any connection of the disease with dead tubercle bacilli. In the first place no microorganism yet known to us can be found within the fresh efflorescences on microscopical examination (*Axenfeld, Wagenmann, Hertel, L. Mueller* and others). Cultures fail likewise to detect ordinary bacteria in these nodes. Dead tubercle bacilli are not to be found in them. In the second place *Leber* and others have demonstrated that the inoculation of fresh efflorescences into the anterior chamber of rabbits excites no inflammation. In the third place the giant cells occasionally found in the efflorescences are not evidences of a tubercular origin, especially as these cells are not of *Langhan's* type. The evidence which has been brought forward does not convince me that keratoconjunctivitis eczematosa is caused by an endogenous infection.

I maintain on the contrary that an ectogenous origin must be granted before the disease can be understood. The superficial situation of the nodules, as well as their discrete character, certainly justifies the theory that morbific agents gain an entrance through the epithelium. In scrofulous persons the epithelium is known to be particularly vulnerable, and we also know that morbific agents exist which are able to penetrate into even an intact cell, such as the parasites of the blood cells.

Many ophthalmologists, like *Axenfeld, Morax, Casparini* and *L. Mueller,* claim that the picture of conjunctivitis eczematosa may be produced in scrofulous individuals by such known agents of conjunctival inflammation as diplobacilli, pneumococci and *Koch-Weeks* bacilli. The only conclusion that can be drawn from such observations is that this disease can be produced by an external infection. We do not know that a specific agent of conjunctivitis eczematosa does not exist in such cases, even though it has not yet been recognized. I question, with *Morax*, whether the eruptions at the limbus met with in *Koch-Weeks* conjunctivitis can be identified with eczematous efflorescences. If an ectogenous origin is admitted there are only two possibilities to be considered. If the efflorescences are caused by any bacteria known to us, it must be by staphylococci, as these are present in eczema of the skin. But, as staphylococci are not to be found in the fresh efflorescences of the conjunctiva, they, or their products, must excite a peculiar reaction with the formation of these discrete efflorescences in the conjunctivæ of scrofulous individuals. I cannot subscribe to this as long as a simpler explanation is possible. The entire clinical picture of keratoconjunctivitis eczematosa becomes comprehensible at once when we assume that a specific agent, as yet unknown, but perhaps widespread, excites the discrete diseases on the surface of the eye, especially in persons with a scrofulous diathesis. This theory cannot be proved correct, but it is rendered plausible by the superficial situation of the efflorescences, the infiltration and the pannus. Every corneal ulcer but this is due to an ectogenous infection; is the eczematous ulcer alone an exception? The secondary infections also harmonize with this theory. Shall we assume in ulcus eczematosum migrans that it alone is the way and manner in which the conjunctiva reacts to irritations of various kinds? I believe that a certain agent plays the part of the unknown x, even when other germs are secondarily present.

II. Course of Keratoconjunctivitis Eczematosa

At the end of a week the efflorescences on the conjunctiva will have healed without injury to the eye, but the moment the cornea becomes involved the prognosis becomes more serious. As long as the lesion remains an infiltration, involution is possible, even though the symptoms of irritation are quite severe and the disease is prolonged, but eczematous ulcers always leave scars. Pannus also is a very serious condition. The worst of the disease is that it recurs so frequently, embitters the happy period of childhood, and casts its shadow over later life by the bad vision caused by so many corneal opacities.

III. Treatment of Keratoconjunctivitis Eczematosa

The first indication in the therapy of this disease of the eye is the general treatment of the scrofulous diathesis. Unfortunately the majority of these cases are met with among the children of poor people, so that only a small part of the programme recommended can be carried out. The measures must be chosen so as to fit each individual case. The statement of *Cornet*, that "air, light, movement and nutrition are the fundamental supports of both the prophylaxis and the treatment," is correct, for we have no specific means at our disposal with which to act upon scrofulosis as such.

The general treatment should begin with the regulation of the usually improper diet, which is frequently lacking in the necessary meat, fresh vegetables and fruit, while the children are surfeited with starchy foods. Too much of any one form of food is to be avoided. The children should be taken regularly out of doors, even when they object on account of their photophobia. An air bath lasting only a few minutes, which may be given in a room, is a stimulant which excites a reaction in the form of an arterial hyperæmia of the body, and is of service in the treatment of scrofulosis. Sun baths cannot be given during the attacks of keratoconjunctivitis eczematosa, because of the photophobia and the pain caused by the direct rays of light, but after the attack has passed away the child should be kept in the sunlight, lightly clothed, playing out of doors; playing on the sea beach particularly is beneficial.

When conditions permit, choice may be made between a sojourn in the country, sea baths, or the mountains. Life in the country sometimes does wonders for city children. The North Sea baths are better for robust children, the Baltic for weakly ones. If high altitudes seem to be indicated, mountains of moderate size and subalpine heights should be chosen.

Saline baths are almost of more importance in the treatment of scrofulosis. Kreuznach, Reichenhall, Rheinfelden in Switzerland, and Duerrheim may be mentioned, but these expensive spas are beyond the reach of most of our patients. Mothers who cannot afford such expense should be taught how to give saline baths at home, by dissolving a sufficient quantity of common salt in the water of the bath to make about a 3% solution. Half salt and half Stassfurt bath salt, or crude salt may be used. When even such measures are not possible, the mother can rub down the child each morning with a cloth wet with salt and water. This is an opportune time to warn against the hardening treatments that are often overdone, and by which delicate children are often injured. Metabolism may be promoted by massage, and suitable gymnastics are of much benefit.

Cod liver oil has been used in scrofulosis since the days of antiquity, still maintains its reputation and is well borne. It is given in doses of a teaspoonful, or a dessertspoonful, from one to three times a day. Many substitutes are on the market, but they have been unable to supplant it. Another remedy that finds much favor is the combination of iodine and iron in the form of the syrupus ferri iodati. When anæmia is a prominent symptom one of the multitudinous preparations of iron should be given. A favorite prescription in childhood is liquor ferri albuminatis in doses of from ½ to 1 teaspoonful. Each physician has to choose which preparation of iron he may prefer, but he should not think that iron in organic combination is absorbed, or utilized by the body any better than inorganic iron, which the physiological chemist *Abderhalden* claims alone is efficient. Arsenic also is used a great deal in the form of liquor potassii arsenitis.

In the treatment of keratoconjunctivitis eczematosa two therapeutic measures quite distinct from the internal and dietetic treatment of the scrofulous diathesis come into consideration: one with regard to the treatment of the diseases of the skin, the other to that of the eye itself.

This patient has an extensive eczema of the scalp, known as seborrhœa. The hair is cut short first, and then the child wears during the night a dressing soaked with olive oil. In the morning the softened crusts are removed, the oil is wiped away with benzin, and sores on the scalp are revealed. An application of a 1% solution of silver nitrate, followed by neutralization with salt and water, will cause these sores to become dry very quickly. After the application the sores are wiped dry with cotton and an ointment is smeared over the entire scalp. Either one of three ointments may be used for this purpose: 1, yellow ointment; 2, zinc ointment; 3, *Neisser's* zinc bismuth ointment, composed of zinc oxid. 1.0, bismuth. subnitr. 1.0, ungt. simpl. and ungt. leniens each 10.0. Vermin, especially lice, frequently cause this eruption; in such cases the children should wear for twenty-four hours a dressing wet with sabadilla vinegar.

In many cases it is well to apply an inunction of ungt. kalii iodat., or ungt. iothion, to the swollen glands.

Examination and treatment of the interior of the nose is necessary in many cases. The eczema of the nares may be easily controlled by the regular introduction of pledgets covered with yellow ointment.

Even when there are no marked lesions in the skin, excellent results are to be obtained from the inunction of the entire body with a mild green soap. A teaspoonful of the soap is rubbed over the body for five or ten minutes twice a week by the nurse, who then places the child in a warm bath and washes it clean. How these inunctions of soap are of benefit in scrofulosis I cannot say, but it is unmistakable that they exert a powerful influence over the metabolism in such children and I can recommend them strongly. We turn now to the treatment of the eye itself, and I wish, first of all, to call attention to a point that is not dealt with even in the most modern textbooks.

Parents of photophobic children with blepharospasm often bind up their eyes in the hope of doing some good in that way. The contrary is frequently the case. Such children are often in a very dirty condition. The lacrymal fluid stagnates beneath the even slightly soiled dressing, or the conjunctival secretion becomes readily decomposed, thus increasing the trouble and the danger to the eye. All dressings are to be avoided in the great majority of cases of keratoconjunctivitis eczematosa. We use them only when the greatest possible quietude of the eye is rendered necessary by the presence of very deep corneal ulcers. Air and light should be allowed to enter these eyes.

Here is a child with an intense blepharospasm who not only resists every attempt to open his eyes for examination, but keeps them closed so tightly that the lids have become swollen in consequence of the interference with circulation. The cause of this blepharospasm must be sought first.

Very often it is produced by the painful sores on the skin at the outer canthus, the rhagades, which, when present, need to be treated first. The mitigated stick of silver nitrate is rubbed over the little sores after the skin has been dried thoroughly with cotton and then, before the skin can become moistened again by the tears, *Lassar's* paste is applied. This paste consists of acid. salicyl. 0.3, amyl. 0.3, zinc oxid. 3.0, vaselin alb. ad 10.0. It should be applied frequently during the day. In these cases the blepharospasm disappears only after the healing of the often obstinate rhagades is complete.

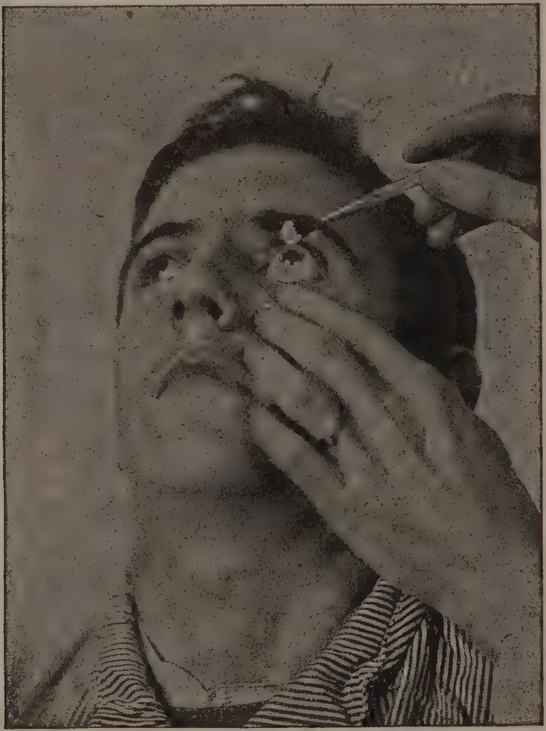


FIG. 25.—Powdering with Calomel.

If no rhagades are present the blepharospasm must be caused by the eczematous efflorescences on the conjunctiva or cornea, and may be stopped as if by magic, at least for a time. If the face of the child is immersed in a basin of cold water until the necessity of breathing begins to be felt, he will open his eyes. When it is possible to distract the attention of the child, the blepharospasm may be obviated for some time, sometimes forever. Nervous influences play a part in this condition.

These simple measures will not always suffice. Sometimes the palpebral fissure is very narrow, a condition known as blepharophimosis, and this renders treatment difficult. Nothing can be accomplished in such cases until the fissure has been enlarged by an operation at the outer canthus.

Here is another type. Although conjunctivitis eczematosa as such is a discrete disease, in which the rest of the conjunctiva is not involved, many cases are met with, especially such as have been neglected, which have a diffuse catarrh with considerable secretion. Such cases are said have scrofulous congestive catarrh. A 1% solution of silver nitrate applied to the lid and then neutralized should commence the treatment. In these cases it must be ascertained also whether blepharospasm exists.

The method of treatment is quite different in the next case which presents as its most prominent feature a deep, eczematous ulcer of the cornea that is threatening to perforate. A bandage should be applied unless there is considerable secretion, in which case the eye is to be cleaned regularly and kept covered with a protective, latticelike eye shield. In other respects the treatment is that ordinarily employed in corneal ulcers. Atropin is employed regularly; it is applied best in the form of an ointment. A salve composed of hydrarg. bichlor. 0.003, vaselin alb. ad 10.0 may be used; it is borne better than any other mercurial ointment. After the ulcer has begun to improve the eye should be massaged regularly with yellow ointment. The use of eserin instead of atropin has been recommended in cases in which the ulcer that is threatening to perforate is peripheral, so as to prevent so great a prolapse of the iris, but I believe this to be based on mere theory. When perforation has taken place the prolapse should be left alone and the eye kept under a protective bandage; after the irritation has subsided completely it may be cauterized, or excised.

When no grave corneal ulcer is present the local treatment of keratoconjunctivitis eczematosa is simpler. The two most important remedies employed locally are calomel and the yellow oxide of mercury.

Calomel is principally used in cases of marginal efflorescences in which the eyes are otherwise unirritated, and when a migrating ulcer is commencing. The head of the child is bent somewhat backward, the lower lid is drawn down from the eyeball, a dry camel's hair pencil is dipped in the powder and then tapped gently with the finger, so as to dust the calomel over the conjunctiva. The powder gathers together into little lumps and the patient closes the irritated eye. The principal action of this remedy is chemical, though it produces a slight mechanical effect. The chemical action set up between it and the salt contained in the tears transforms a portion of it into sublimate, but in what its final action consists in keratitis eczematosa still passes our knowledge. There seems to me to be no doubt that it is the mercury, or its combinations, which exerts such a beneficial influence in this disease. Can any conclusion as to the nature of the morbific agent be drawn from this? A part of the mercury locally applied to the eye is excreted in the urine.

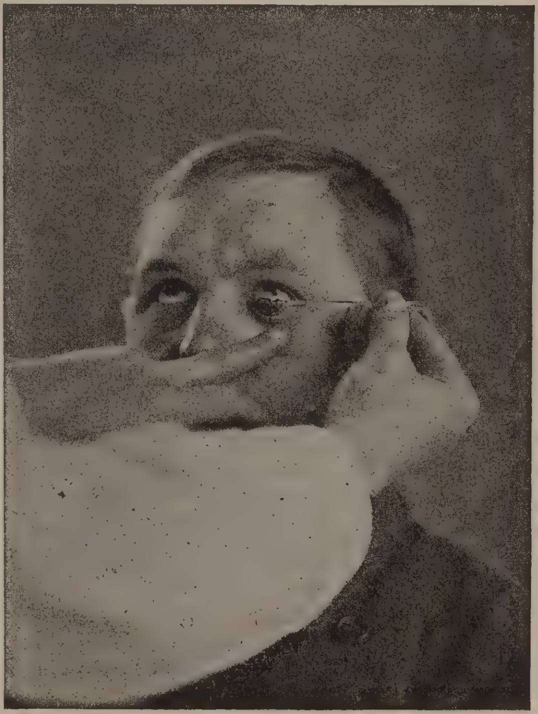


FIG. 26.—Application of Yellow Ointment.

Hydrarg. oxid. flav. via humida parat., yellow oxide of mercury, is used in the form of the yellow ointment, known also as *Pagenstecher's* ointment, which is indispensable in ophthalmology. There has been much dispute concerning its preparation. The yellow oxide of mercury must be distributed very finely, the fatty excipient must not be rancid, and it must be introduced properly into the eye. The ointment as recommended by *Schanz* and prepared by *Schweisinger* consists of hydrarg. oxid. flav. via hum. par. 0.1, adeps lanæ, aqu. destil. ā ā 1.0, vaselin alb. ad 10.0. Its range of application is greater than that of calomel. Efflorescences in the conjunctiva and at the limbus, pannus eczematosus, ulcers in the stage of regeneration and small infiltrates are massaged with it twice a day. Patients must learn to use it themselves, for to obtain good results in keratoconjunctivitis eczematosa it is essential that it should be used for months, sometimes for years. It is also the most efficient means at our disposal for the clearing up of corneal opacities. To apply the ointment the lids are separated a little, some of the salve is taken on the end of a clean glass rod and placed on the inner surface of the lower lid; the lids are then closed and the rod is withdrawn from between them, leaving the ointment in the conjunctival sac. A piece of cotton is then placed on the closed lids and the salve is distributed over the surface of the eye by massage with the fingers. Patients should be told to massage the eye in this way while they are counting twenty or thirty. The application should be repeated once or twice a day. The strength of the ointment may be gradually increased, but not above 2% for the patient's own use. Frequently it irritates the entire conjunctiva and produces a marked hyperæmia which, in conjunction with the effect of the mercury, exerts a beneficial influence. The attacks may be arrested and recovery brought about, in the majority of the uncomplicated cases, with the aid of these two remedies. Migrating ulcers are apt to be obstinate, especially when the infiltrated head is very thick. Such cases should be treated as soon as possible with punctate cauterization.

33. Xerosis of the Conjunctiva and Keratomalacia

We comprehend under the term xerosis all of the changes in the cornea and conjunctiva which result in a markedly dry condition of this otherwise wet and lustrous organ. It may be due to disturbances of the general nutrition in which the cornea is affected together with other parts of the body, or it may be the consequence of local causes. The latter are divided into two distinct groups, one comprising the cases in which the cornea becomes dry because the lids close imperfectly, the other those in which the xerosis is the result of cicatrization in the conjunctiva although the lids close properly. *Sæmisch* called the form that results from cicatrization of the conjunctiva xerosis parenchymatosa, the other forms xerosis epithelialis. Xerosis parenchymatosa is irreparable, xerosis epithelialis is capable of improvement and cure.

I. Xerosis due to Nutritive Disturbances Throughout the Organism

I have here a child less than a month old, who has had cholera infantum for some time and is now in the last stage of marasmus. He is extremely emaciated, his skin is pale, his features almost aged, his voice thin and feeble. When the eyes are opened we find in one that the cornea has lost its beautiful lustre, looks dull and dry, is covered with scales and crusts, is opaque and no longer sensitive. The conjunctiva below and on each side of the cornea is dry and covered with whitish masses that give it a peculiarly livid, dirty appearance. When the child moves his dead dull eyes the conjunctiva bulbi forms crescentic folds on the side toward which the cornea turns. In the other eye the condition is farther advanced. Yesterday the cornea was yellowish gray, especially in the central part, today it is purulent and perforation has taken place. The iris is prolapsed. Sometimes the lens is extruded. This is the picture of infantile keratomalacia. I show the case for three reasons.

In these cases the body is weakened excessively by the profuse diarrhœa and we can readily agree with *Foerster* that the nonvascular tissue of the cornea fails to obtain sufficient nutrition. It is also probable that in such a marasmus abnormal products of decomposition circulate in the blood, and are responsible for the reduction in sensibility and the loss by the tissues of their power of resistance. At any rate it can be conceived that the xerosis is caused by these disturbances of nutrition and toxic influences. *Foerster* has appropriately compared this condition with that of bed sores. In addition to this the eyes of such children are apt to be incompletely closed during sleep, so that the portion of the cornea which is exposed is allowed to get dry, but this necrobiosis of the cornea and conjunctiva may take place even when the lids close well.

Participation by secondary infections is to be added to the disturbance in nutrition. Xerosis bacilli were found in great abundance in xerosis by *Kuschbert* and *Neisser*, and were taken by many to be the morbific agents, but they are present in the healthy conjunctiva and are to be considered as harmless parasites. They are not the pathological agents of xerosis and keratomalacia.

When suppuration and ulceration occur in xerosis and keratomalacia other pus agents, such as staphylococci and streptococci, have acted upon the weakened and unresisting cornea. I can show you a slide from a bad case of keratomalacia in an adult, who had lived for years in a horribly poor condition of nutrition, in whom both corneæ melted away in a few days. It contains bacilli belonging to the group of bacterium fusiforme and an extraordinary number of spirochætæ. Aside from the spirochæta pallida these are the first spirochætæ that have been demonstrated in the human cornea.

This example demonstrates that keratomalacia is not simply one of the associated symptoms of cholera infantum. It is met with in children suffering from grave congenital syphilis, when it need not be a specific syphilitic keratitis. It has been met with in other infectious diseases, such as dysentery, typhus, puerperal fever and scarlet fever.

The most important reason why I have exhibited this child with cholera infantum and keratomalacia is to show the prognostic importance of the condition. The prognosis quoad vitam is very grave. Even when children are not so far gone as this one, yet have xerosis and keratomalacia, the prognosis is very bad, especially when the child is quite young. In such cases an attempt must be made to increase the strength through the treatment of the cholera infantum; strengthening diet, mucilaginous soups, weak meat broths, calomel, iced milk; hot baths when signs of collapse appear. I have obtained good results in many cases of keratomalacia from the subcutaneous injection of large quantities of salt solution. As far as the eye is concerned we must be content to bathe it carefully and to apply ointments.

The Benign Form of Xerosis Conjunctivæ

The form of xerosis just described, which is one of the symptoms of keratomalacia, may be styled the malignant form of epithelial xerosis. Another form, likewise due to general nutritive disturbances, is much more benign. It appears as a symptom of, or in connection with, a form of hemeralopia.

There are conditions in which patients complain that they see very badly as soon as twilight begins; these will be considered under diseases of the retina. This hemeralopia may be associated with certain pathological changes in the retina, or it may be a functional disturbance without visible ophthalmoscopic lesions, due to nutritive disturbances throughout the entire organism. A xerosis of the conjunctiva is met with in the latter form. Insufficient nutrition may compromise the functions of the retina. In Russia great numbers of people are affected epidemically during the seven weeks' fast before Easter. This form of xerosis is met with in convicts, children in orphan asylums, and sailors, everywhere that the food is deficient and without variety. Peculiar, dull, lustreless places appear on each side of the cornea in the palpebral space, which look as though smeared with fat and have a characteristic triangular form, with their bases toward the cornea, their apices toward the canthi. *Bitot* was the first to observe these spots in cases of hemeralopia, so they were named *Bitot's* spots. They are found on both eyes. Adults are generally affected, though the condition may be met with in children, coincidently with the night-blindness. *Uhthoff* called attention to the fact that both hemeralopia and xerosis conjunctivæ may occur in chronic alcoholism. Both symptoms are also met with in chronic liver diseases; it is well to remember this when an epithelial xerosis of obscure origin is met with.

Finally a dryness of the cornea has been observed in cholera, which is to be ascribed not only to the lagophthalmos in the stage of collapse, but also to the loss of water by the entire body and the disturbance of nutrition.

This hemeralopia also occurs with and without coincident xerosis in people who are apparently healthy; in these cases we know nothing of the nature of the trouble. I have repeatedly examined such persons serologically without finding anything positive.

The prognosis of this form of xerosis is much better, it passes away with the improvement in the general nutrition. Liver has been recommended as a curative in hemeralopia. How great a part nutrition plays is shown by a case reported by *Uhthoff*. A student, who was a fanatical vegetarian, became so reduced in strength that he began to suffer from xerosis and hemeralopia; the hemeralopia disappeared in four days after he began to take eggs and milk.

II. Xerosis of the Cornea from Local Causes

A. Xerosis Due to Imperfect Closure of the Lids

In our next patient the closure of the lids is imperfect because of a facial paresis. The lower lid, particularly its inner portion, is not raised properly, but is turned from the eyeball; ectropion paralyticum. Consequently there is epiphora. The lower part of the cornea is imperfectly covered during sleep and the result of this is not only an irritation of the conjunctiva, but also a superficial dullness and dryness of the cornea with a loss of its sensibility. This condition is the so called keratitis e lagophthalmo.

Other causes besides facial paresis produce such a xerosis of the cornea. When an eyeball is pushed far forward in exophthalmos the closure of the lids may be prevented and the surface of the cornea may become dry. Some time ago I had a patient a large part of whose upper lid had been torn off by the kick of a horse. During sleep his cornea had been without sufficient covering and partial xerosis had begun. In all of these cases we should try to correct the imperfect closure of the lids, either by shortening the palpebral fissure, or by a plastic operation, but much good can be done by having the patient wear a moist dressing during the night, and by the use of an indifferent boric acid ointment.

B. Xerosis of the Conjunctiva and Cornea after Cicatrization

We have next an unfortunate trachoma patient with a third form of xerosis. The saddest end of malignant trachoma is an extensive dryness of the conjunctiva and cornea. The palpebral fissure is narrow, the conjunctival sac is very small, the entire conjunctiva looks as if it were studded with whitish scales and elsewhere smeared with fat.

This form of xerosis differs from the others in an important feature: it is the result of a local cause, the cicatricial contraction of the trachomatous conjunctiva. Formerly it was thought that this process began with an occlusion of the efferent ducts of the lacrymal gland, and this theory seemed plausible, especially because such patients complain very early of the feeling of dryness, but it is not correct. The feeling of dryness is due to the fact that the lacrymal fluid can no longer affect the compact, shrunken conjunctiva with its thickened epithelium. The cicatricial contraction of the conjunctiva is the primary cause, an atrophy of the lacrymal gland follows later.

The same grave xerotic condition may be produced by the cicatrizations caused by pemphigus conjunctivæ, diphtheria and burns. In pemphigus the shrunken conjunctiva is whitish, cloudy and dry. In very bad cases even the lacrymal secretion fails, because the emunctory ducts of the lacrymal gland have become occluded.

In all of these cases we must be content with a symptomatic treatment; ointments may be introduced into the eye before going to sleep, milk may be instilled during the day to alleviate the symptoms; physiological salt solution, or a 1% solution of sodium bicarb. may also be employed. *Nessnamoff* had such patients smell of oil of mustard and aqua ammonia several times a day, in the hope that a hypertrophy of the lacrymal gland might be obtained thereby. Attempts have been made to replace the shrunken membrane by grafts of healthy conjunctiva. Sometimes the lids have been sutured together leaving only a small aperture. Whoever has to treat unfortunate patients tormented by xerophthalmos must try many things in the hope of giving comfort.

34. The Most Important Varieties of Corneal Ulcers and their Sequelae

The diagnosis of a corneal ulcer is so important that we will devote a chapter to a consideration of some of the questions connected therewith. The recognition of its clinical peculiarities often reveals immediately what disease is present in the eye. Corneal ulcers heal through the formation of connective tissue and therefore leave opacities which interfere with vision, hence it is very important that they should receive proper treatment.

The forms of corneal ulcer most commonly met with in practice are 1, ulcus serpens; 2, ulcus eczematosum; 3, the marginal ulcer frequently called ulcus catarrhale. More rarely we meet with 4, the diplobacillus ulcer and 5, the trachomatous ulcer. Even though the true cause cannot be proved in perhaps the majority of cases, or some other etiology excluded, except by the bacteriological demonstration of the morbific agent, yet we are able usually to distinguish the different varieties by their purely clinical characteristics.

The ulcus serpens is a disease of middle and old age. It invariably follows slight injuries, appears almost always in the central part of the cornea and spreads over its surface. It is associated with secondary iritis and hypopyon.

An analogous clinical picture may be produced, not by pneumococci, but by diplobacilli. Like ulcus serpens the diplobacillus ulcer is met with in adults, and is situated usually in the central part of the cornea. The differentiation can be made only by the bacteriological examination.

The ulcus eczematosum is very easily recognized clinically. It is one of the symptoms of keratoconjunctivitis eczematosa, a disease of childhood, and is therefore met with in children. It differs from ulcus serpens in its roundish form, as well as in its tendency not to spread over the surface, but to penetrate into the parenchyma and to perforate the cornea. It may be found anywhere on the cornea, at its margin as well as at its centre, and is accompanied by the other symptoms of keratoconjunctivitis eczematosa, photophobia, blepharospasm, rhagades at the canthi and the symptoms of scrofulosis.

The ulcers of the cornea met with in the course of catarrhal inflammations of the conjunctiva are just as typical and easily recognized. As indicated by the name these ulcers lie near the margin, and they have a characteristic crescentic form. During the course of a conjunctivitis a row of little infiltrates appears at the limbus, extend, become confluent, throw off the epithelium and the most superficial layer of the cornea, and form a long, sickle shaped ulcer, which deepens rapidly and may perforate. Sometimes several such ulcers appear in an eye and they may surround the cornea.

The ulcer met with in trachoma can be recognized readily from the presence of other signs of that disease, particularly of pannus trachomatosus. The ulcer is horizontally oval, and situated near the centre of the cornea on the line of demarcation of the pannus. Thus we can and must determine clinically, as soon as we see an ulcer of the cornea, what kind of an ulcer it is.

The treatment depends largely on the stage in which we find it. When the ulcer is progressive the eye has a marked ciliary injection, the base and margins are gray or yellowish, from the infiltration of pus, and a hyperæmia of the iris, or an iritis, can be perceived. When it is regressive the margin is melted away and the base is smooth and polished.

In a fresh catarrhal ulcer every effort must be made to control the conjunctivitis as quickly as possible. Silver nitrate solution should be applied energetically, the eye kept washed out and all dressings abandoned which may cause a stasis of secretion. In eczematous ulcers the keratoconjunctivitis is to be treated generally and locally, atropin and sublimate ointment are to be employed and a bandage is to be avoided so far as possible. The principles of the treatment of ulcus serpens have been discussed already. The treatment of trachomatous ulcers must be directed primarily against the trachoma.

We do not always see these ulcers in their early stages; often we have to deal with their sequelæ and from these we have to draw conclusions with regard to the preceding ulcerations. A study of these sequelæ facilitates the understanding of the conjunctival and corneal diseases, for their nature and their anatomical changes are always the same, whether they come from a primary ulcer of the cornea, or from one that is secondary to a conjunctival disease. *Cicatrization* in the cornea is the first consequence under all circumstances. All *leucomata* and *maculæ* are results of corneal ulcers.

This man has a dense, central leucoma which covers the pupil. No vision can be given the eye except by means of an optic iridectomy. This operation differs from an iridectomy for glaucoma; it cannot be made upward and it should be small so as to make the dazzling as slight as possible. In order to ascertain whether an optic iridectomy is likely to be of benefit in such a case, the vision is tested, then the pupil is dilated and the vision tested again. Sometimes a decided improvement can be obtained in this manner, but the retinal images are never sharply defined when the light enters through the peripheral, less uniformly curved portion of the cornea. The opacity on the cornea aids in the production of dazzling and for this reason, as well as for the sake of the cosmetic appearance, it should be tattooed.

Thus far we have dealt with the normal course of recovery of a corneal ulcer. Sometimes the cicatrization stops before the loss of substance has been quite replaced and leaves a concave, lustrous surface. Such places are called *corneal facets.* Or a thin, transparent cicatrix may yield before the intraocular tension and rise above the surface of the cornea, forming a *simple ectatic corneal cicatrix.* Conditions become complicated when a scar following the perforation of an ulcer becomes ectatic.

In another patient a minute, rather convex bleb, surrounded by a light gray margin, can be seen in a loss of substance, which is elsewhere lustrous, near the centre of the cornea. This is a *Descemetocele,* or keratocele. At this place the ulcer destroyed the parenchyma down to *Descemet's* membrane, which was able to resist its action, and is now distended and pushed forward by the intraocular pressure. In such a case the patient must be put to bed and a firm bandage should be applied. Healing takes place through the development of connective tissue, which constantly grows firmer, about the little bleb. The bleb may burst, allow the aqueous to escape, and so permit the iris and lens to move forward. Healing may still be obtained in most cases by careful cauterization, when the anterior chamber will be reformed, but a little dark point will remain visible for a long time. A *corneal fistula* may be developed in such a place. This may open and close repeatedly, and keep the eye constantly soft, so that it may be rendered blind by detachment of the retina, unless the fistula can be induced to heal. If seen in time such a fistula may be closed by covering it with conjunctiva in the way recommended by *Kuhnt.*

In most cases *perforation of a corneal ulcer* takes place without having been preceded for any length of time by a keratocele. The signs of perforation are easily recognized. The patient himself feels the escape of the stream of warm aqueous, the tension of the eye is decreased, the anterior chamber is abolished, the iris and lens have moved forward. It is remarkable how much benefit the ulcer receives from the perforation; evidently the nutrition of the tissue is improved by the reduced tension. What follows depends on the size and situation of the perforation.

An eye that has suffered from a perforating ulcer of the cornea can be recognized for many years. The pupil is distorted, pear shaped and drawn to a point; the anterior chamber is deeper at one place than at another. Both of these symptoms can be referred to a little blackish mass near the inner margin of the cornea of this patient, where the iris is adherent to the posterior surface of the cornea and forms an *anterior synechia.* The point of the pyriform pupil is at this place, and here the anterior chamber is either shallow, or abolished. An anterior synechia may be produced also by a penetrating wound, when the iris is caught between its lips and becomes adherent. In this case the round form of the black spot makes it extremely probable that a round, eczematous ulcer perforated the cornea at this point during childhood. When the cicatrix composed of iris and cornea is of large extent it is called a *leucoma adhærens.*

We may have to deal therapeutically with a leucoma adhærens after a long time has elapsed. When the surface over such a cicatrix, that contains both iris and cornea, becomes ulcerated a malignant infection may penetrate rapidly into the eye by way of the iris. Sometimes also it is advisable to divide the iris from the synechia for visual, or cosmetic reasons. Still more frequently such an anterior synechia induces secondary glaucoma by a partial occlusion of the angle of the anterior chamber.

An early stage of this condition may be seen in this child with keratitis eczematosa. A peripheral ulcer has perforated and the anterior chamber is incompletely restored. The most striking symptom presented is a black brown knob, larger than the head of a pin, which is raised above the surface of the cornea. This is a fresh *prolapse of the iris.* As the aqueous escaped the iris was drawn into and more or less through the opening, where it readily became agglutinated to the inflamed tissue about the edge of the ulcer. The color of a prolapse varies; small ones are dark, but when the iris has been stretched a good deal it approaches a gray. The color also depends on the length of time the prolapse has existed, for an incarcerated iris that lies exposed to the air between the purulent margins of an ulcer soon undergoes further changes. The inflammation quickly seizes upon this portion of the iris, covers it with a gray layer of exudate and soon transforms it into a granulating tissue. Then the color is reddish gray. By degrees brighter gray spots of connective tissue appear in it. If conditions are favorable, and it is not too large, the prolapse gradually flattens in consequence of the contraction and fusion of these cicatricial areas, until finally nothing is left of the original prolapse except a scar evenly level with the cornea, through which the remains of the iris tissue shine darkly and with which the iris remaining in the anterior chamber is directly connected. The picture now presents the condition seen in the previous patient.

Two deviations from this normal course of healing of a prolapse of the iris are met with, one of which is to be seen in grave cases of ulcus serpens. When the contraction of the cicatricial tissue in an extensive corneal ulcer is particularly great the entire cornea may be flattened. This is called *applanatio corneæ.* Usually a prolapse of the iris is present in one place or another, though applanatio may occur in cases in which a large portion of the cornea has been destroyed down only to *Descemet's* membrane. The cicatricial cornea also can undergo a high degree of contraction and produce what we call *phthisis corneæ.*

In this case the cicatrix formed from the remains of cornea and prolapsed iris has not become flattened, but ectatic; such an ectatic cicatrix of iris and cornea is called a *staphyloma corneæ.* The cornea is replaced by an opaque, hemispherical, protuberant cicatrix with an overhanging margin sharply differentiated from the sclera. The pigment of the iris shines through, giving it a bluish appearance; the color is slate gray and black in places. It has received the name staphyloma because of its resemblance to a blue grape. Large bloodvessels coming from the conjunctiva are to be seen in it and it is evidently opaque.

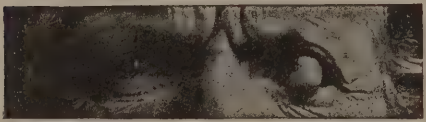


FIG. 27.—Staphyloma Corneæ Totale.

This is a total staphyloma of the cornea. Sometimes only a portion of the cornea is involved, when we have a partial staphyloma, the form of which is usually not hemispherical, but conical (staphyloma conicum). At first the wall of a staphyloma is very thin; but it gradually consolidates and dense cicatricial bands may be formed, which constrict it more or less, and give it the appearance of a staphyloma racemosum. In old staphylomata the wall is thick and white, with some dark spots. It is self evident that no anterior chamber exists in a case of total staphyloma, but it is otherwise with partial staphylomata, which are usually situated somewhere at the margin of the cornea. Then the tensely stretched iris is visible through the transparent cornea, and the further progress of the staphyloma may be checked by an iridectomy.

A staphyloma is understood to begin in this way: After the perforation has occurred the iris prolapses as the aqueous escapes, continues to protrude and, together with the remains of the cornea, is transformed into cicatricial tissue. The larger the original perforation, the larger becomes the staphyloma.

The consequences produced by the formation of a staphyloma can be seen in this patient. Evidently vision is destroyed and we may be thankful when even perception of light remains. In partial staphylomata alone a portion of the vision may be preserved, or restored by an iridectomy. Aside from the disfigurement it produces, a staphyloma is a source of irritation to the eye. If the protrusion is so great as to be insufficiently covered by the lids, its surface becomes dry and finally ulcerates. Worst of all it increases the tension by the displacement of the angle of the anterior chamber. As the tension increases glaucomatous pains commence and the last degree of vision dies out along with the development of a glaucomatous excavation of the optic nerve. The increased tension drives the wall of the staphyloma still farther forward and makes it thinner; it may rupture, collapse and refill, until finally an intraocular infection terminates the process by an atrophy of the eyeball. In young persons the sclera may yield and give rise to an intercalar, or ciliary staphyloma.

These facts justify us in attempts to prevent the formation of a staphyloma whenever possible. An iridectomy should be performed immediately to counteract the increase of tension as soon as a corneal ulcer shows a tendency to protrude, but this can be done only when a portion of the anterior chamber is present. Nothing can be done in a case of total staphyloma except to remove it. When the staphyloma is very great and the entire eyeball is enlarged as well, enucleation is the best procedure, but when a stump can be preserved the prothesis will fit better and be more movable. In such a case it may be excised in the way described by *Beer*.

The conjunctiva about the limbus is dissected up, a few sutures are inserted in it with which later to close the wound, it is then retracted, when the lower part of the staphyloma is abscised by a curved incision, the flap is seized with forceps and the upper part removed with scissors. The lens is then allowed to escape and the wound is closed by means of the sutures.

If the perforation takes place in the region of the pupil it does not become plugged by a prolapse of the iris and the anterior chamber remains empty for a longer time. The lens is pushed forward until its anterior surface is in apposition with the ulcer; the inflammation causes the epithelium of the anterior capsule to proliferate and so gives rise to an *anterior polar cataract.* Afterward the ulcer heals, the lens falls back with the iris as the anterior chamber is reformed, but the polar cataract remains a permanent evidence that the eye has suffered from a corneal ulcer, although the corneal opacity itself may clear up wonderfully. Sometimes such a cataracta polaris anterior may be produced without perforation of the ulcer by the diffusion of the toxic products into the anterior chamber, where they cause the epithelium of the anterior capsule to proliferate.

Another complication, met with in cases of blennorrhœa, is the *luxation of the lens.* When the perforation of the ulcer takes place under high pressure, e.g., when the child is crying and squeezing its eye, the lens may be displaced forward suddenly, the fibres of the zonule may be ruptured and the lens is then found between the margins of the lids.

A rare consequence of the perforation of a corneal ulcer is a grave *intraocular hemorrhage.* If the chorioidal, or retinal vessels of the patient are arteriosclerotic the sudden loss of tension produced by the perforation may cause them to rupture, and allow large quantities of blood to escape into the eye. The hemorrhages may be so great as to press the entire contents of the eyeball through the aperture. This happens sometimes in cases of absolute glaucoma in which the perforation of a corneal ulcer takes place. A pressure bandage, absolute rest in bed and exenteration form the entire treatment.

Although perforation usually exerts a favorable influence upon the course of the ulcer itself, yet it is sometimes followed by an extension of the infection into the eye and the production of *panophthalmitis.* This happens in cases of grave blennorrhœa and ulcus serpens.

These are at least the most important sequelæ of corneal ulcers. They inflict a greater or less loss of vision every year upon thousands; a sufficient reason why the patients, and the members of their families, should have their attention directed to them, and that proper treatment of the corneal ulcers should be urged.

35. Clinical Picture of the Pneumococcus Ulcer (Ulcus Serpens) and its Social Aspect

The various causes that may produce corneal ulcers need to be studied in connection with the clinical pictures they inaugurate. More or less of the cornea may be destroyed by a burn of the eye with an acid or alkali; when the necrosed portion is thrown off an ulcer remains, the origin of which is revealed by the history. Much more often the ulcers met with are the results of infection and are of greater practical importance.

We have here an old man whose eye was slightly injured a few days ago by a twig of a tree; the injury was so slight that at first it scarcely attracted his attention. A day or two later he began to suffer from severe pain in the eye, especially at night, and now a fresh ulcer is to be seen in the cornea.

In order to ascertain the cause of this ulcer a bit of material is taken from its margin, with a strong platinum needle, after the eye has been cocainized, smoothed out on a cover glass, stained with dilute carbolfuchsin and then examined under the microscope. Numerous lancet shaped diplococci are thus found about the pus cells, a few within them. This microorganism, the *Frænkel-Weichselbaum* diplococcus lanceolatus, is the ultimate cause of a definite form of corneal ulcer, the ulcus corneæ serpens. This ulcer is met with in middle and old age and always is preceded by an injury. It is not met with in childhood, although pneumococci are found frequently in the corneal ulcers of children. The people who suffer from ulcus serpens belong, almost without exception, to the working classes; farmers, reapers, etc. The trouble always follows an injury, but the traumatism may be so slight as to escape the notice of, and even be denied by the patient.

In the majority of cases signs of chronic conjunctival catarrh at least may be found, and very often a disease is present which is of serious importance in such superficial injuries of the cornea, dacryocystitis. If pressure over the region of the lacrymal sac produces regurgitation of a mucous or purulent secretion, the physician must consider the danger that an ulcus serpens may develop when a superficial injury occurs to the cornea. If such an ulcer already exists the physician turns instinctively to the lacrymal sac. Dacryocystitis is present in certainly more than half the cases of ulcus serpens. Strictures of the lacrymal passage may exist even when there is no profuse flow of pus from the sac; these will be found the more frequently as they are sought for more carefully.

The examination for coincident lacrymal disease in a case of ulcus serpens is of great importance, because this is the chief source of supply of the agents that provoke the ulcer. A slide prepared from the pus of the lacrymal sac shows it to swarm with pneumococci. It must never be thought that these agents were introduced by the foreign body that inflicted the injury, they invariably enter the injured place as a secondary infection. A lesion of the epithelium of the cornea always must exist before these germs can secure a foothold in the living tissue.

The intact, healthy epithelium is impermeable to pus agents and forms an efficient protection to the eye, even when that organ is inundated day after day for years by thousands of these germs, when dacryocystitis is present.

Even when there is no dacryocystitis, pneumococci find their way into wounds of the cornea. They are met with not rarely in chronically inflamed conjunctivæ. Many patients have the bad habit of washing their eyes with saliva, which nearly always contains pneumococci when the mouth is not properly cared for. Abundant opportunities occur for infection among these people and ulcus serpens is a fairly common disease. In many eye clinics it forms five or six per cent of the clinical material. The bacteriological investigations made by *Uhthoff*, *Axenfeld* and others have demonstrated that, in the overwhelming majority of cases, ulcus serpens is caused by the diplococcus lanceolatus, the same germ which is the agent in croupous pneumonia. I myself obtained pure cultures of pneumococci in 95 out of 100 cases of ulcus serpens in the eye clinic at Wuerzburg.

Mild and grave cases are met with, as in every disease. The severity of an attack depends on the general susceptibility, the local power of resistance in the cornea, the nature and severity of the injury and, by no means least, on the virulence of the pneumococci present.

Each ulcer begins with an infiltration which, under favorable conditions, may proceed no further; in such cases we speak of a pneumococcal infiltration, not of an ulcer.

As soon as a more serious infection takes place severe pains are felt in the eye and head, that are aggravated at night. The cornea about the defect in the epithelium becomes cloudy, and a yellowish white infiltrate appears in one or more places, usually in the margin of the defect. In a later stage the ulcer presents itself as a gray white, or yellowish gray, disclike opacity in the central portion of the cornea and cannot be mistaken. The reason why the centre of the cornea is the part regularly attacked is because it is the most prominent portion and therefore the most apt to be injured. The cloudiness is more marked at the margin than in the middle of the ulcer. Usually the infiltration of pus is greatest on one side, and indicates the direction in which the ulcer tends to spread; this place has been termed the edge of progression and contains great masses of pneumococci.

In order to understand correctly the clinical picture of ulcus serpens some of the details of the fight made by the eye against the invading microbes need to be recognized. With the aid of a loupe it can be seen that the ulcer is surrounded to a great extent by a delicate gray areola, and that fine, radiating, gray striæ extend from its margin far into the clear cornea. These fine striæ are formed of wandering leucocytes, which come from the marginal network at the limbus and press toward the focus of infection. They have been called leucocyte paths (Leucocytenstrassen).

The symptoms in the anterior chamber and iris are very marked. An accumulation of pus, a hypopyon, forms at the bottom of the former. The latter is discolored, its details are indistinct, the pupil is contracted and an exudate is formed at its margin which is productive of posterior synechiæ. One characteristic of ulcus serpens is that the entire pupillary portion of the iris appears to be peculiarly and uniformly reddened. In other words, every grave, freshly formed ulcus serpens is accompanied by an iritis, the origin of which should be understood.

The toxic products of the infection are not confined to the cornea, but penetrate by diffusion into the anterior chamber, in the same way as atropin. In the anterior chamber they have a good opportunity to excite an inflammatory hyperæmia of the iris, as well as a contraction of the pupil, by their action upon the bloodvessels of the iris and ciliary body. If the toxic irritation is sufficient pus cells emerge from these vessels toward the focus of infection, the aqueous becomes more and more cloudy, the pus cells cling to the posterior surface of the cornea, or sink to the bottom of the aqueous, an excretion of fibrin is added and the hypopyon is formed. Thus a secondary toxic iritis is excited by the primary focus of infection in the cornea. As the ulcer is superficial, at first destroying only the outermost layers of the cornea, and as *Descemet's* membrane is a very resistant tissue which does not permit the passage of cells, the hypopyon remains composed of sterile pus, free from pneumococci, as long as no perforation takes place. When the latter happens the infection naturally may extend into the eye, and pneumococci may be found in the anterior chamber and even in the vitreous.

The clinical pictures of this disease vary from the minute pneumococcal infiltration, which heals smoothly and leaves scarcely a visible trace, to the complete destruction of the cornea, with final suppuration of the contents of the eyeball, in proportion to the malignancy of the microorganisms and the power of resistance of the tissues. The cases may be divided into three groups, the mild, the moderately severe, and the gravest forms of ulcus serpens.

Whatever may be the course of healing in the individual cases the results are always the same; cicatrization in the cornea. These results may be illustrated by a few cases.

In the first case may be seen a central, comparatively transparent cloud over the pupil, which may properly be termed a macula. There are some adhesions of the iris to the lens capsule (posterior synechiæ) and the vision is much impaired. In the second case the consequences are much worse. A large part of the cornea is occupied by a white leucoma which leaves only a narrow margin of clear tissue. The superficial and middle layers of the cornea were destroyed over a large area and the loss of substance thus produced was filled in with new vascular tissue. Large bloodvessels may be seen passing through the leucoma. The sight of such a case as this leaves the indelible impression that ulcus serpens is one of the most dangerous diseases of the eye.

So much stress might not be laid on the clinical study of this disease if it did not have an important social aspect. It is a disease of the laboring classes, it always destroys the central part of the cornea which is necessary to vision, it follows slight injuries and is therefore classed as an accident; these facts explain its great social importance, a conception of which may be gained from this table.

ULCUS SERPENS IN THE AGRICULTURE AND FORESTRY ASSOCIATION OF UNTERFRANKEN.

Year.	Number of Certified Injuries of the Eye.	Number of Ulcera Serpentia.	Percentage of Ulcus Serpens.	Year.	Number of Certified Injuries of the Eye.	Number of Ulcera Serpentia.	Percentage of Ulcus Serpens.
1889..	3	0	0.0	1896..	29	11	37.9
1890..	7	2	28.5	1897..	38	24	63.1
1891..	16	3	18.7	1898..	60	29	48.3
1892..	11	2	18.1	1899..	45	30	66.6
1893..	12	7	58.3	1900..	50	26	52.0
1894..	17	12	70.5	1901..	55	33	60.0
1895..	29	11	37.9				

This table presents the material collected in the Agriculture and Forestry Association of Unterfranken, of which I formerly had charge, under an agreement with the kingdom of Bavaria, so far as it relates to ulcus serpens. It shows that the reported injuries of the eye have increased from year to year, an increase which is chargeable directly to this disease. From the first there was no doubt in the minds of the members of the association that grave injuries of the eye, whether perforating or contused, entitled them to the insurance benefit. But after a few years, after the purpose of the accident insurance law became understood more widely, and the fact became generally known that an ulcus serpens follows a slight injury, the sufferers learned to claim indemnity for trivial injuries as well. In 1889, when the law was passed, there was not a single ulcus serpens among the three reported injuries of the eye. In 1890, 1891 and 1892 it formed only 28.5 and 18.7% of the injuries of the eye in this association. From 1893 on the number of ulcera serpentia reported as accidents increases rapidly and constitutes at least one half of the reported injuries of the eye. The majority of these persons draw their indemnities permanently. The official statement of the government for Wuerzburg is that from the time of the passage of the law to December 31, 1901, a period of thirteen years, 35,000 marks had been paid by this association alone to persons who had suffered from ulcus serpens. When it is taken into account that there are in Germany forty four such agricultural and forestry associations, and that there are forty nine other associations connected with this industry, it is evident that the total accident indemnity for ulcus serpens is an enormous amount, even though the conditions vary in different associations and in different regions. To this must be added the expense incurred in the nursing and care of these cases, as well as the loss of wage earning power, and of the pleasures of life incurred by the victims of this injury, in order to obtain an adequate conception of the great importance of this disease to the family, the community and the State. This must continue to increase unless an advance is made in its prophylaxis.

36. Therapy of Ulcus Serpens and the Duty of the General Practitioner in its Prophylaxis

We have now two problems to solve: first to learn to treat this disease itself; second to consider how far and within what limits the general practitioner can take part in the prophylaxis of this corneal infection which is productive of such serious consequences.

I. Treatment of Ulcus Serpens

The therapy of ulcus serpens is a problem on which our science is constantly at work, so, in order to inform ourselves concerning all the factors concerned, we proceed in this clinic in the following manner.

After cocainization of the eye a bit of tissue is taken from the progressive margin of the ulcer with a flat needle, prepared on a cover glass and examined to determine whether the pneumococcus is the morbific agent. This is necessary, because a similar clinical picture may be produced by diplobacilli. If pneumococci are present cultures are made from the margin of the ulcer. Then before treatment is commenced two questions are to be considered:

First, whether a dacryocystitis is present. If there is a purulent condition of the lacrymal sac containing pneumococci, the treatment of the ulcer is begun indirectly by the extirpation of the sac, for it is not quite a matter of indifference to the course of the ulcer whether or not its surface is constantly inundated by thousands and thousands of these pathogenic germs. This purulent dacryocystitis is present in more than 60% of the cases of ulcus serpens.

If the lacrymal sac has been removed, or if no dacryocystitis is present, the choice of treatment depends on the character of the ulcer, which may be very malignant, moderately severe, or mild.

In mild cases the process may cease with a little pneumococcal infiltration, or with an ulcer having a fine, gray wall, which may heal in a few days. In the moderately severe cases a progressive tendency is unmistakable, but the symptoms of irritation and the distant effects upon the iris are comparatively slight, while the progression is gradual. In malignant cases the inflammatory symptoms are very severe; there is a marked chemosis of the conjunctiva and a dense, broad, yellow, purulent wall at the margin of the ulcer in the cornea, which advances rapidly, and constantly starts new foci in the deeper layers. An ulcer may appear to be not so very malignant at first and then suddenly spread. Secondary infections with staphylococci are not uncommon in large, malignant ulcera serpentia. The general practitioner cannot decide what form of treatment is indicated because he lacks the necessary experience; *it is therefore his duty to send his cases of ulcus serpens to the clinic, or to the ophthalmologist without delay.* When this is impossible he may act along the following lines.

In a case clinically so malignant that it is to be feared that the advance of the ulcer cannot be checked, the progressive margin should be cauterized immediately with the galvanic cautery, in order to destroy the morbific germs and arrest the process. In addition sublimate ointment, or an antiseptic powder, like iodoform or airol, may be applied to the surface of the ulcer, but these means are scarcely ever able to kill the morbific agents in the living tissues; their destruction is attained much better by cauterization, which at present is the sovereign remedy in the treatment of ulcus serpens.

Sæmisch recommended a free incision across the ulcer. A narrow knife with its edge directed forward is introduced at the margin of the cornea, passed from one limbus to the other, and the cornea is cut across through the centre of the ulcer. The aqueous escapes and, according to his directions, the wound should be reopened several times with a stylet. There is no doubt that recovery is promoted in many cases by this artificial perforation, just as benefit is obtained from spontaneous perforation, yet I decline to perform this operation. Very often the iris prolapses between the lips of the wound and forms a leucoma adhærens which may produce further trouble, and, even when the result is good, a scar is left that passes through the middle of the cornea and creates conditions not favorable to the success of a subsequent optic iridectomy, which must be made in the palpebral fissure.

Grave cases of ulcus serpens may be treated by paracentesis, when a reduction of the tension by an artificial opening of the anterior chamber is indicated. A keratome is introduced at the limbus into the anterior chamber and the aqueous is allowed to escape slowly. If the knife is withdrawn too quickly the sudden fall of tension may induce a hemorrhage, especially in old people with atheromatous bloodvessels. Such a paracentesis is necessary in many cases immediately after the cauterization, or subsequently, when the hypopyon is large, viscid and cannot be absorbed readily. In such cases the thick, coagulated lumps of pus must be removed with forceps through the wound. Frequently it is not until after the removal of such a hypopyon that it can be seen distinctly how corroded is the base of the ulcer, or how great is its extent. After the ulcer has been cauterized and the hypopyon removed, atropin is instilled, the eye is bandaged and the patient placed in bed. Atropin must be used freely in all cases of ulcus serpens to counteract the iritis and the formation of posterior synechiæ. Thus far the treatment of a grave ulcus serpens is simple and clearly indicated. Most ophthalmologists rightly consider this radical cauterization the surest form of treatment; it certainly far surpasses all methods of purely conservative treatment with antiseptics. I believe this, and yet I believe that cauterization cannot permanently remain the ultima ratio of the therapy of this ulcer. According to my conception of the nature of the process of infection and of its healing, it seems to me to be going too far to cauterize all ulcera serpentia. We should rather try to avoid cauterization whenever a radical operation is not absolutely necessary. This proposition is supported by the following clinical facts.

First, it happens not infrequently that cauterization performed once, twice, or three times does not accomplish its purpose, because certain pneumococci escape and new generations gain a foothold in the necrotic areas.

Second, and I want to emphasize this point, there are cases in which the condition grows worse after the cauterization.

Third, it cannot be disputed that the cautery destroys not only the cocci, but also tissue of the cornea which contains only exudate and is still capable of recovery. The destruction, or at least vital injury to the function, of perfectly healthy corneal tissue cannot be avoided in many cases if the cauterization is thorough, no matter how carefully it may be performed.

Fourth, some ulcera serpentia recover spontaneously without progression; in these cauterization is unnecessary.

Fifth, there is no doubt that the corneal opacities which remain after cauterization are more extensive and more opaque than those left when the disease is overcome by the living tissue. This undisputed fact is proven not only by clinical observation on the human eye; I have demonstrated it with exactitude by animal experiment.

Sixth, the collective results of the treatment of ulcus serpens up to the present time, in which cauterization is the principal remedy, make any attempt to gain even a slight advance seem desirable. This is acknowledged even by ophthalmologists who regard serum therapy with little favor. These reasons justify the inquiry whether we *need* to cauterize immediately ulcera serpentia which do not *demand* immediate cauterization. The question is how to try to arrest the infection and promote spontaneous healing.

I have based the serum therapy of ulcus serpens on the knowledge that in bacterial infections an advance in treatment is to be sought only in the specific etiological therapy. We introduce into the body of the person suffering from a pneumococcal infection of the eye specific antibodies, in the expectation that these will counteract the infection and so render it easier for the body to overcome the disease. This proposition, which a priori seems reasonable, rests upon the fact, first established by *Lœffler*, that the eye, and particularly the cornea, partakes in the immunity of the entire organism. I have tested this biological law repeatedly, on the broadest lines, for all infective agents. Without exception it is proven that the antibodies are able to penetrate into the nonvascular cornea in general active and passive immunization. The opinion which many hold that the cornea does not participate in the immunity produced by vaccination is incorrect. This immunity is the one in which the participation of the cornea was demonstrated with great exactitude by my assistant *Grueter*. Like every serum therapy this one has its limits.

The limits of serum therapy are narrower for corneal diseases than for infections of other organs. This is inherent in the metabolism of the cornea, in the fact that the antibodies can penetrate only to a moderate degree and slowly into the tissue. If these limits of serum therapy in the cornea are borne in mind only moderate results will be expected from it in ulcus serpens, and its value will not be overestimated. It is announced, with a certain degree of satisfaction, that the serum treatment cannot supplant the old, radical, operative treatment of ulcus serpens. I have never expected that it could. Such a demand is not reasonable; it is not even just. There is no therapy in ophthalmology which can completely supplant other therapeutic measures. This is true of all operative and medicamental procedures. If so great a demand is made on the serum therapy of ulcus serpens a priori as the criterion for its right to exist, the slight advantage thus obtained may be too easily lost. Our therapeutic knowledge advances but slowly, and we should be thankful for a little gain. *This gain, which in my opinion is obtained through the specific serum treatment of ulcus serpens, is the healing of quite a number of ulcers which otherwise would have had to be cauterized.* I consider this an advance because in these cases the conditions are more favorable for the clearing up of the corneal opacity than after cauterization. In what cases the serum therapy can accomplish this purpose, and in how large a percentage it is sufficient, has not yet been decided. The result depends on the malignancy of the case, the dosage, the nature of the serum and the species of the pneumococci.

It is very difficult to determine positively that the serum was really of use when healing has followed its administration, though certain clinical signs are of assistance. During the first and second days after the injection of the serum there is a strong local reaction at the margin of the ulcer and then the progressive edge melts away quite rapidly. To obtain a better criterion from which to judge whether a favorable turn of the disease after the use of serum is due to this therapy, a culture of pneumococci is made from the ulcer and this stock is tested, first, to determine its virulence, second, to ascertain whether the serum is able to overcome an infection with it in the body of an animal.

A dose of serum is injected into a number of white mice; twenty four hours later these, together with a number of controls, are infected with varying quantities of the pneumococcal culture. In this way a useful indication of the virulence of the stock is obtained, while at the same time the experiment proves whether antibodies are present in the serum employed, which can specifically arrest the action of the stock from the ulcus serpens in question. It has been learned from these experiments that the stocks of pneumococci in ulcera serpentia vary greatly in virulence. Generally speaking, the more malignant the ulcer, the more virulent is the stock of pneumococci.

A comparison of the virulence of stocks taken from ulcers the spontaneous healing of which may be awaited, and from those in which cauterization is necessary, with that of stocks taken from ulcers that have recovered under serum treatment goes to show that serum may accomplish its purpose in cases which otherwise would not have healed spontaneously. In support of the clinical observation the conclusion may be drawn from this, at least with some probability, that when such an ulcer heals under serum treatment the addition of the antibodies was not wholly in vain, and that the immunization of the body aided the healing in a manner analogous to that ascertained to take place in animals by experimental infections of the cornea. This is our criterion by which to judge what part serum plays in the treatment of ulcus serpens. We have none better, and ophthalmology has the advantage over other specialties that the lesions are so plainly to be seen. But the actual conditions of serum therapy are more complicated, for the parallelism between the virulence of the pneumococci in animals and the clinical course of an ulcus serpens is not mathematical. This indeed is not to be expected, because in all diseases the susceptibility of the individual, as well as the virulence of the pathogenic agent, affects the entire course. Hence a very virulent stock may be taken from an ulcer which runs a benign course, or a malignant ulcer may contain much less virulent pneumococci. All these experiments serve to answer the one question, whether it is probable that serum can be of use in many cases. It can no longer be denied that *there are cases in which a curative action of the pneumococcus serum is exhibited very distinctly.* Clinical observation has confirmed that which at first I demonstrated experimentally: the eye partakes of the general immunity of the organism and the cornea is not excluded from this participation because of its lack of blood-vessels. The questions whether a successful result can be anticipated in any given case, and whether the cases fitted for serum therapy can be selected by purely clinical observation, must still be answered in the negative. It is certainly easy to treat only mild cases with serum, but spontaneous recovery occurs and no mild case that recovers after the use of serum can be said to have been cured by the remedy. The curative substances produced in the bodies of animals follow fixed pharmacodynamic laws. On the other hand I believe, with the majority of the advocates of immunity, that a lack of comprehension of the action of these substances is shown when specific therapy is completely thrown aside because it fails in grave cases. The golden mean is evident to any competent judge. The stand I take is: to try the serum therapy in every case in which the malignancy does not necessitate an immediate cauterization, because spontaneous healing affords a better chance to the eye than cauterization. If the ulcer threatens to spread in spite of the serum treatment it is to be cauterized immediately. I am under the impression that even then the repeated cauterizations are fewer in number, and that in other respects the conditions for recovery are better in many cases, but this is not certain as yet. If the ulcer does not incline to spread I adhere to the serum treatment.

It is true that a large expense has been incurred in vain in the cases in which cauterization has to be performed eventually, for the serum is not cheap, but I believe that a clinic should provide these means.

When the case is one in which the attempt to use serum therapy may be made without detriment to the eye 50 ccm of the pneumococcus serum are injected beneath the skin of the abdomen of the patient. It has been demonstrated that the antibodies are more concentrated in the antitoxic than in any other form of the serums that act antagonistically to infectious bacteria, and it has been proved that only a comparatively slight and transient concentration of the antibodies is obtained in the human body, even when large quantities of the serum are used, because of the dilution it undergoes. It has been proved with regard to diphtheria serum that large quantities must be used to obtain its curative power, and this is applicable even more to the other serums. It is commonly known that this way of using serum is harmless and even the totally unfounded fear of anaphylaxis is gradually disappearing. Under certain conditions the small doses of serum are of value, but therapeutic results are not to be expected from them. Pneumococcus serum therefore is to be given in doses of from 25 to 50 ccm because smaller ones are ineffectual. After the injection the patient is put to bed and serum free from carbolic acid is dropped on the ulcer several times a day in order to bring the antibodies into direct contact with the diseased tissue. The local use of the serum alone is insufficient, as I have demonstrated experimentally. A transient increase of the hypopyon does not always indicate progression, it may occur because more toxic products have been set free by an increased mortality among the bacteria. The guiding factor is the behavior of the progressive margin of the ulcer. This may be increased by a local reaction, but when it exhibits no tendency to melt away at the end of twenty four or forty eight hours, or when other signs of a true progression are present, such as the formation of new foci, cauterization should be delayed no longer. When signs of improvement appear the injection should be repeated on the second or third day. By that time it is certain whether anything can be hoped for from this form of treatment in that particular case. We can wait somewhat longer for deep infiltrations which do not spread in the base of an ulcer, because they require a longer time for absorption and the cautery must be passed deeply into the tissue in order to reach them. As the founder of this specific serum therapy for ulcus serpens I am free from utopian ideas and prejudice, but I maintain that the little progress fortunately secured by this form of treatment should not be cast aside. It would be a grave injury to our science and to our patients if we should weary of the only rational therapy, that directed against the etiology of the disease in the way indicated by the study of immunity. Whoever expects a miracle from the word serum is under an illusion. If we use the specific antibodies in selected cases of pneumococcal inflammation of the eyes and are sufficiently alert to discontinue the experiment in time, whenever necessary, without injury to the eye, we have done all that science demands from the newly established point of view. A single eye saved in this way with a faultless cornea compensates amply for the failures. It is self evident from the difficulties that beset the specific etiological therapy that a nonspecific serum therapy is hopeless in ulcus serpens. This fact was to have been expected from the results of the study of immunity, and has been demonstrated by *Axenfeld* and his student *Happe*. There is no scientific foundation for the idea that such nonspecific serums as diphtheria serum and *Deutschmann's* yeast serum can possibly produce any effect upon a pneumococcal infection of the cornea.

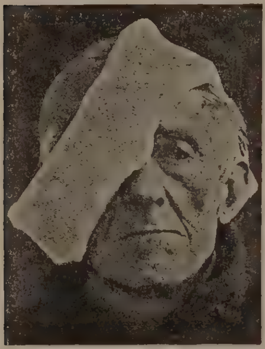


FIG. 28.—Thermophor on the Right Eye.

When an ulcer is in its final stage, when the progress of the infection has been checked, the necrotic, infiltrated parts of the margin have been cast off and the base has become clean, the object of therapeusis is to aid the processes of healing in filling up the defect. Hot, moist compresses with our thermophor are applied to the eye for hours at a time.

Frequently the final results of an ulcus serpens also demand therapeusis. When the ulcer was not very malignant, or was arrested in so early a stage that the corneal opacity left seems to be capable of becoming more transparent, the eye should be massaged with yellow ointment for weeks and months. In young persons the opacity sometimes becomes remarkably small and clear in the course of the first year. When such an event may be expected, and the case is one which has been reported as an accidental injury, the association should be advised to have a reexamination made at the end of a year. After no further increase in transparency can be hoped for operative measures often are necessary.

In some cases cosmetic reasons seem to demand that the white scar in the centre of the cornea should be tattooed. This operation is often performed in combination with an optic iridectomy. When the centre of the cornea is occupied by a dense, opaque leucoma, while a part remains clear at the periphery, a slight amount of vision may be restored by this operation, which allows the light to enter the eye through the peripheral portion of the cornea and lens.

Other operations that are necessary at times are exenteration, when the ulcer has perforated and the vitreous has become purulent, or an iridectomy to reduce increased tension after the ulcer has run its course, or an operation for staphyloma.

II. The Duty of the General Practitioner in the Prophylaxis of Ulcus Serpens

The more we study ulcus serpens the stronger grows the impression that this is one of the most dangerous diseases of the eye, that it is one fraught with the most serious consequences. Therefore every effort should be made to promote prophylaxis, which can be attained only step by step through the education of generations of physicians. There are two ways in which the general practitioner can render very efficient aid.

The most important source of infection in ulcus serpens is dacryocystitis. In the majority of cases the pneumococci which infect the small, superficial wounds of the cornea come from the lacrymal sac, and as we have learned already, a spontaneous recovery from dacryocystitis cannot be expected among the laboring classes. Many cases of ulcus serpens could have been avoided by the timely extirpation of the diseased lacrymal sac. The most important prophylactic measure which can be undertaken is the early performance of this operation, introduced as a means of prophylaxis by *Axenfeld.* It can be performed under cocain anæsthesia and is absolutely without danger. Advise all patients, especially if they belong to the laboring classes, to have a suppurating lacrymal sac extirpated. By doing so many an eye can be guarded against ulcus serpens.

The second duty of the general practitioner in the prophylaxis of ulcus serpens is not to undertake the treatment of a case himself, but to send it immediately to the specialist.

I always strive to define the limits within which a general practitioner can practice ophthalmology, and I believe that I make these limits broader than many, but ulcus serpens is not a disease the treatment of which should be entrusted to any one who has not had the training of a specialist. The bad results today, as seen by a review of the entire material of a clinic, depend in great part on the fact that the patients arrive too late. To illustrate this fact I append this table of forty cases of ulcus serpens treated in my clinic, showing the number of days that had elapsed between the receipt of the injury and the admission.

Day after the Injury.	Number of Cases.	Percentage of the Total Number.	Day after the Injury.	Number of Cases.	Percentage of the Total Number.
1	0	0	8	7	17.5
2	2	5	9	0	0
3	5	12.5	10	2	5
4	4	10	11	0	0
5	2	5	12	3	7.5
6	0	0	13–16	10	25
7	2	5	17th day to 3 weeks	3	7.5

These statistics tell a sad story. Twenty-five per cent. of all the cases came between the second and the fifth days, 25% more between the seventh and the tenth, and 25% more between the thirteenth and sixteenth days after the receipt of the injury. I know very well that this is due in a great many cases to the negligence of the patients, but in not a small number better results might have been obtained if they had received timely and energetic advice to place themselves at once under the care of a specialist. Still there is one thing that I think the general practitioner can do, particularly in the country, whence most of these cases come. As no time should be lost, and only too frequently valuable time is lost before the patient can reach the specialist, I advise the physician to examine a slide prepared from the margin of the ulcer and, if pneumococci are found, to administer a prophylactic injection of pneumococcus serum. The earlier the disease is recognized the more urgent, in my opinion, is the indication for such a prophylactic injection, for there is no doubt that the prophylactic effect of a specific immunization always gives a better prospect of success than a therapeutic use of the same remedy at a later stage. This law of immunity applies to the serum therapy of corneal infections and has been tested in the cases of all pathological agents met with in the eye. In an infection of the cornea with virulent pneumococci, furnished with thick capsules, the most favorable time for a specific serum therapy is past in a few hours. Such virulent, so called serum fast, generations of pneumococci, which are little accessible to the antibodies, can originate during the course of the infection. The earlier they are counteracted by the specific antibodies the more favorable a priori will be the conditions. One fact is unfortunate. There are stocks of pneumococci against which the serum is ineffective; this is true of all kinds of antiinfectious serums. This fact is taken into account in the manufacture of serums and a large number of stocks is always used in making the serums provided for immunization. It is therefore possible that the prophylactic injection of serum may prove to be ineffective in any particular case, but this can never be known in advance. Our results would be still better if we could use homologous serum, but this is impossible and we have to be content with coagulated horse serum.

The prophylactic extirpation of the lacrymal sac in dacryocystitis, the sending of a case of ulcus serpens immediately to the specialist, and the prophylactic injection of serum after a positive diagnosis of a pneumococcal infection has been made, constitute all that it is in the power of the general practitioner to accomplish. We will be thankful for such assistance, and so will be the patients, for prophylaxis is the most important point in the fight against ulcus serpens.

37. The Diplobacillus Ulcer of the Cornea

At the first glance this ulcer appears to be quite similar to ulcus serpens. It is situated almost in the centre of the cornea, is fairly extensive, is disc shaped, and has its margin distinctly more infiltrated on one side. It is also accompanied by an iritis and a fibrino purulent exudate at the bottom of the anterior chamber, which may be called a hypopyon.

Yet this is not an ulcus serpens. The expert can frequently distinguish it by its clinical appearance. An ulcus serpens of this size usually shows signs of depuration in a certain place, while in this ulcer the infiltration is about the same in all places and the base seems to be much more deeply infiltrated than is common in ulcus serpens. The processes of progression and depuration do not go hand in hand in this ulcer so much as they do in the other. After the ulcer has healed symptoms of irritation persist in the eye for a considerable length of time. But its etiology can be determined positively only by a bacteriological examination. The agent of the ulcus serpens is the pneumococcus; the agent in this disease is the *Morax-Axenfeld* diplobacillus. The origin and course of these ulcers are analogous to those of ulcus serpens. A slight erosion of the epithelium of the cornea permits the entrance of diplobacilli, the chemotactic action of which produces infiltration and suppuration; an ulcer is formed which spreads and may reach a considerable size.

The processes of healing in these ulcers are very interesting. Their malignancy is not so very great in comparison with that of ulcus serpens, even when the case appears to be serious. We meet with signs of bacteriolysis, as well as of phagocytosis, in their spontaneous healing. I have found bactericide material in human blood serum which acts energetically upon these bacilli, and this furnishes an explanation why these ulcers heal under the treatment we employ.

We know from experience that these ulcers heal with greater difficulty after they have been cauterized. The bacteria multiply again very rapidly in the burned portion and therefore the adjacent corneal tissue should be injured as little as possible by the treatment. Our purpose is attained best, according to the experience of *Axenfeld*, *Erdmann*, *McNab*, and others, by the use of zinc, just as in diplobacillus conjunctivitis. The zinc should be used frequently, dropped directly on the surface of the ulcer and applied at night in the form of an ointment. It has been shown already, in the study of diplobacillus conjunctivitis, that the zinc does not directly kill the microorganisms, so we must conclude that the remedy acts through a change produced in the metabolism and indirectly aids the tissue in its fight with these bacteria. Still we meet with cases that do not recover quickly under the influence of zinc, and with others that recover spontaneously without it. To instill the drops the head of the patient is bent somewhat backward and the conjunctival sac is filled with a weak solution of zinc several times during the day, sometimes every hour. In the evening zinc ointment is applied to the ulcer.

I have dwelt on this ulcer for three reasons: to demonstrate that the diplobacillus is able not only to excite a conjunctivitis, but also to injure the cornea, to show how the curative influence of the remedy employed is intelligible, and to impress the necessity of making a positive diagnosis by the bacteriological examination of slides in every case in which ulcus serpens is suspected and in which the patient cannot be sent immediately into the clinic. In case of certainty pneumococcus serum may be injected, but such an injection would be useless in a diplobacillus ulcer. The general practitioner, when in position to make such prophylactic injections, must learn and be guided by the etiology.

There are other infections of the cornea which, though rare, should be mentioned. A keratitis is met with which is caused by infection with *mold fungi*. A circular opacity forms about, and at a little distance from, the central focus, in which the foreign body that inoculated the spores may sometimes be found. The central ulcerated portion presents a peculiarly dry, rough appearance and is finally sloughed off. There are also mild cases of mold fungus keratitis that resemble migratory ulcers. All of these are produced by the aspergillus fumigatus. The effect of this fungus upon the cornea has been studied particularly by *Leber* in his able work on the origin of inflammation. In very rare cases of hypopyon keratitis *Stoewer* and *Lundsgaard* found a rose colored yeast (Rosahefe). I have also a slide showing bacteria first found by *zur Nedden* in a number of infectious marginal ulcers.

38. Keratitis Parenchymatosa

We come now to the study of a disease of the cornea which often is not recognized at the decisive moment, yet is of great interest, not only to the specialist, but to the general practitioner as well, because it is an evidence of a general disease of the organism. The most important points in the clinical picture may be pointed out in this patient and afterward we may look at several others.

The ciliary injection preponderates over the conjunctival. The cornea appears to be uniformly cloudy, its surface dull and without lustre. The vagueness of the opacity indicates that it is situated deeply in the tissue. By means of *Zeiss'* loupe the cloudiness may be resolved into separate maculæ, none of which are superficial. The deep situation of the opacities is proven positively by the minute, tufted vessels which can be traced between them and disappear at the limbus. An inflammation like this in the deeper layers of the cornea is denominated a keratitis parenchymatosa, or interstitialis.

The clinical course in a typical case is as follows: Associated with increasing symptoms of irritation in the eye, sometimes with only a slight ciliary injection, a place at the margin of the cornea loses its lustre and becomes cloudy. By means of the loupe this cloudiness can be seen to be composed of little cloudy maculæ. After a while similar opacities appear in other places at the limbus; these gradually become confluent with the first and then the central portion is gradually encroached upon, until finally the entire cornea has a blue gray appearance. The epithelium is minutely dotted so as to give the cornea an appearance as if it were smeared with fat. The time required for the cornea to become completely cloudy varies very much. It depends on the case. Sometimes the course is tempestuous, sometimes weeks and months elapse before the cornea has become quite opaque.

As soon as, and very often before, the disease has reached this stage peculiar changes are noticeable. The limbus looks red and swollen and the process of deep vascularization of the cornea begins. Beneath the swollen limbus minute vessels grow forward into the middle and deeper layers of the cornea, run parallel with each other and present a penicillate, or tufted, branching. The most marked clinical feature of these vessels is that they disappear at the limbus, which is explained by the fact that they come from loops of the perforating scleral branches of the anterior ciliary vessels beneath the marginal network. These peculiarities suffice to differentiate this vascularization from that met with in pannus. The vessels in parenchymatous keratitis can only be seen indistinctly and appear to be of a gray red color, because they are covered by a layer of cloudy cornea. Both the opacity and the vascularization vary extremely in intensity. There are cases in which vessels appear only in certain sectors and in which only a portion of the cornea is affected by the opacity.

At the height of the disease the cornea may be so opaque that the iris can scarcely be seen through it, it may be of an almost brown red color in consequence of the great vascularization, and it may be absolutely without lustre. It is self evident that the vision is badly impaired in this stage.

This disease is not an affection of the cornea alone. The name keratitis parenchymatosa has been given it because the principal symptoms are recognizable in the cornea, but, as it involves more or less of the entire anterior segment of the eyeball, it may properly be named uveitis anterior. At the commencement of the disease, and as long as it can be seen well, the iris is it least very hyperæmic and the pupil inclines to contract. Frequently there is a true inflammation of the uveal tract; synechiæ are formed, atropin must be used freely, and deposits often appear on the posterior surface of the cornea. The involvement of the uvea is shown above all in the fluctuations of the intraocular tension, which is lowered at times and at other times elevated.

After an interstitial keratitis has run its course and the cornea has become transparent again, the involvement of the uvea can still be recognized very often with the ophthalmoscope. Characteristic changes may be seen in the anterior portion of the chorioid, especially when hereditary syphilis was the cause of the disease. Little reddish yellow, brownish, or black spots may be seen, situated for the most part in front of the equator; or there may be little white spots, joined together perhaps in clusters. These signs of hereditary syphilis are chiefly in the chorioid and probably depend on changes in the vessels. These symptoms are convincing that the cornea is not alone involved in an interstitial keratitis.

In a typical case the course lasts for weeks before the inflammation reaches its height, and then begins to retrogress. The cornea gradually grows more transparent between the deep vessels at the margin, while the vessels themselves become fewer in number. The centre remains opaque longer than the rest, but by degrees the clouds here are scattered also. One of the most important characteristics of this disease is that in the majority of cases the cornea is able to clear up to an extraordinary degree; often it is difficult to find a trace of the opacity, but here and there a delicate, diffused cloudiness may be detected. A very important clinical fact is that ulceration of the cornea never occurs in this disease.

Yet, throughout life, if the pupil is dilated and light is thrown into the eye with the mirror of a loupe, an examiner, when the focus is right, can recognize many of the old vascular tracts left by the condition that formerly existed and so obtain positive proof that the eye has suffered from interstitial keratitis. Usually these traces of vessels can be seen with *Zeiss'* loupe.

The prognosis of this disease is good in the majority of cases, in spite of its long duration, because of this wonderful clearing up of the cornea. Patients are apt to become discouraged by the slow course of the disease and sometimes wander from one physician to another. Their courage should be sustained by the prospect of eventual improvement. Dense, aponeurotic opacities persist, with applanatio corneæ, or an ectasis of the softened cornea, only in very grave cases.

Thus far the course of a typical case has been portrayed. The difficulty in the recognition of the disease depends chiefly on the existence of a number of deviations from what may be called the legitimate course.

The interstitial inflammation occurs sometimes almost entirely without vascularization, although a careful examination with the corneal microscope nearly always will detect some vessels. In this form of the disease the opacity exhibits a predilection for the centre of the cornea and finally breaks up into little clouds after a prolonged duration.

In other cases the trouble is confined to a single sector of the cornea, and in still other cases the interstitial keratitis starts with a diffuse, rapidly increasing opacity of the whole cornea.

Separate, deep infiltrations may lie near each other for a long time without becoming confluent. This form, which is particularly obstinate, is called keratitis interstitialis punctata. Similar clinical pictures in the cornea may be met with in the iridocyclitis associated with acquired syphilis.

We also meet with cases of parenchymatous keratitis in which the entire opacity is confined to the lowermost part of the cornea, where it assumes a triangular form with the apex directed upward. This picture must not be mistaken for that form of corneal opacity which appears in the lower segment when an exudate in the angle of the anterior chamber has laid for a considerable time in contact with the cornea.

It also happens in the course of an otherwise typical parenchymatous keratitis that the infiltrates in the centre of the cornea unite in the form of a ring, leaving the centre transparent for some length of time, until finally this picture is blotted out and replaced by a central opacity. This form was named by *Vossius* keratitis annularis.

Parenchymatous keratitis is a disease of youth. The patients are usually between the ages of four and twenty years, the average age being from twelve to fourteen years. In very rare cases it has been found at birth and it is occasionally met with between the ages of twenty five and thirty five.

The most frequent cause is hereditary syphilis. Percentages vary in different places, but in my experience congenital syphilis has been present in over 70% of the cases. Acquired syphilis likewise can cause interstitial keratitis, a fact that should be borne in mind when the patient is no longer youthful.

The disease which, next to syphilis, is the most common cause of parenchymatous keratitis is tuberculosis. The clinical course in this form is quite analogous to that in the syphilitic, except that it is rarely so acute and that it frequently occurs in only one eye.

The dense infiltration of the posterior layers of the cornea, with the newly formed bloodvessels, may be seen plainly on the slide, which also furnishes anatomical proof that the inflammation is not confined to the cornea, as the infiltration can be seen to extend from its margin to the iris and ciliary body. Little heaps of round cells, precipitates, lie on *Descemet's* membrane. In many cases the uveitis preponderates over the corneal changes. Since the discovery that the spirochæta pallida is the morbific agent of syphilis these changes in the cornea have been the subject of much experimental study.

None but a very uncritical observer would claim that the therapy of parenchymatous keratitis is satisfactory. We cannot guard against the occurrence of the disease in the second eye. When hereditary syphilis is the cause inunctions of mercury may be given, or iodide of potassium may be administered, but usually the keratitis takes its course in spite of all our efforts. This is because mercury cannot act efficiently in this nonvascular tissue and probably also because the spirochætæ have become mercury fast. The disease often runs a milder course in the second eye, but whoever has seen many cases has observed the contrary. In adults intramuscular injections of the salts of mercury may be tried in addition to the inunctions; in children the internal administration of pills of mercuric chloride has been recommended. When the *Wassermann* test is negative, and there are no clinical signs of syphilis, a tuberculin test should be made to ascertain whether tuberculosis is present. Then tuberculin treatment may be tried. Good nourishing food and care form a very important therapeutic factor in both hereditary syphilis and other anæmic conditions.

Whatever the etiology may be, the local therapy of the eye remains the same. Atropin is instilled regularly to counteract the iritis; the affected eye is bandaged and much heat is applied during the early stages, when the condition of irritation is greatest. This is recommended as directly beneficial in interstitial keratitis. When the disease has entered upon the stage of retrogression the eye should be massaged with yellow ointment, or with an ointment containing mercuric chloride, or potassic iodide. Dionin may be used, or salt solutions may be injected beneath the conjunctiva. Although we cannot ordinarily curb the disease during its period of progression there is no doubt that during its retrogression very much can be done to assist in the clearing up of the corneal opacities. It is in this stage, which lasts for months, that the general practitioner finds much to do.

Before going farther let us review the diagnosis of this disease, which is sometimes very difficult. When an inflammation of the cornea seems to be confined to the deeper layers two points must be considered: First, it must be determined positively not to be a superficial, but in reality an interstitial inflammation. The peculiar slow course, the infiltration of the cornea to a certain maximum and its subsequent retrogression, as well as the fact that it never results in ulceration, are characteristic features. At its commencement the means already described must be employed to differentiate deep from superficial opacities.

Second, it must be remembered that a parenchymatous keratitis is always the consequence of another general disease. The physician must not be content with the examination of the eyes, the ophthalmic must go hand in hand with the general examination, and, as hereditary syphilis is the most frequent cause of this form of keratitis, the symptoms of this disease must be sought for. The most important signs of hereditary syphilis are:

A history given by the parents of miscarriages, or of great mortality among their children. This history, which excites suspicion, should be obtained very tactfully in order not to excite serious reproaches between the parents concerning the cause of the disease in their child.

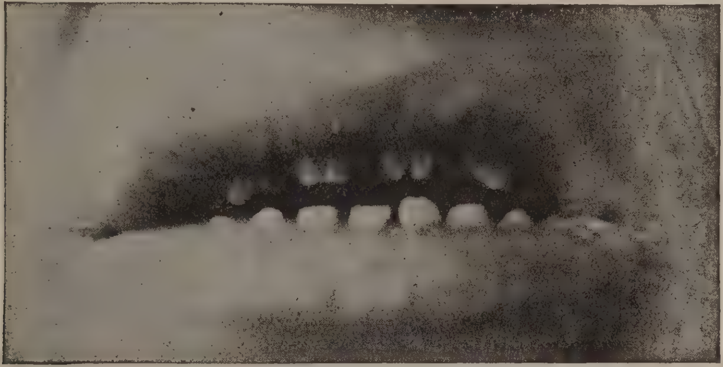


FIG. 29.—Hutchinson's Teeth.

Objectively, the peculiar formation of the face and skull, the protrusion of the frontal prominences, the depression of the bridge of the nose and the flatness of the superior maxilla. About the mouth may be seen fine, whitish, cicatricial bands, in the mouth may be found a smooth atrophy of the base of the tongue and cicatrices in the hard and soft palate, all traces of former ulcerations.

A change in the teeth that has long been looked upon as pathognomonic, if not absolutely demonstrative of hereditary syphilis is the deformation of the two middle, upper incisors, known as *Hutchinson's* teeth. They are imperfectly developed, their lateral margins are not parallel, but convergent downward, and their free margins are indented in the form of a crescent which constantly becomes more excavated. The teeth are also widely separated.

Another important symptom is deafness, which is often due to a disease of the labyrinth, or of the auditory centres, but may be caused by otitis media. The combination of this deafness with the peculiar deformity of the teeth and interstitial keratitis forms the so called *Hutchinson's* triad.

Frequently we meet with an ozæna syphilitica, or a hard swelling of the cervical glands. A swelling of both knee joints may precede, or occur simultaneously with the parenchymatous keratitis. Distentions of the periosteum of the long bones may be present. It must not be expected that all of these signs of hereditary syphilis invariably accompany this form of keratitis; on the contrary this disease sometimes is very difficult to recognize clinically and recourse must be had to *Wassermann's* reaction in order to make the diagnosis. This is particularly necessary when the cause of the corneal inflammation is not evidently syphilitic. Then the diagnostic tuberculin test may be tried. In children this should be done very carefully with small doses. A cutaneous inoculation, as recommended by *v. Pirquet,* perhaps may be made first.

39. The Clearing Up of Corneal Opacities

In view of the fact that corneal opacities so very often impair the visual power of the eye, every effort should be made to clear them up as much as possible. Nature has shown the way to accomplish this within certain limits. The degree to which a corneal opacity is capable of clearing up spontaneously depends, aside from the individual circumstances which must not be overlooked, principally upon three factors.

First, age. Experience teaches that opacities in the cornea do not clear up as quickly and satisfactorily in elderly, as in young people.

Second, the thickness of the opaque tissue. The deeper a scar extends into a cornea the less it can clear up. Perforating cicatrices always remain visible, while superficial opacities may become extremely transparent.

Third, the nature of the opacity and its vascularity. It is conceivable that opacities formed of cellular, or fibrinous exudates may be absorbed, as happens in interstitial keratitis. In such inflammatory opacities the only question is how much the corneal tissue itself has suffered. The conditions are quite different when the opacity consists of a dense, tendinous cicatricial tissue.

In all these metamorphoses of involution the vessels are preeminently the means by which inflammatory opacities are removed from the cornea. The first light places appear in their neighborhood in many cases, but that they are not alone in the possession of this teleological importance is self evident. In the study of corneal diseases we constantly meet with processes by means of which the organism, or the tissue of the cornea, endeavors to clear up these opacities, and the problem for us to solve is how to aid them therapeutically.

In order to obtain a survey of the whole subject we will consider first what can be done in the clinic, and then the part to be taken by the general practitioner.

The idea naturally suggests itself to replace the opaque tissue by the transplantation of transparent cornea in suitable cases. An operation for this purpose is known as *keratoplasty.* My assistant *Loehlein* has obtained an excellent result through the following method.

A fork shaped instrument having on each prong a very small, semicircular knife provided with a guard, is drawn without pressure over the cornea from the upper to the lower limbus. The parallel incisions thus made do not perforate the cornea, but form the lateral boundaries of the flap to be removed. A short conjunctival flap is dissected up above and below and then the rectangular corneal flap, together with both conjunctival flaps, is removed by means of a rounded cataract knife, leaving the posterior layer of the cornea intact. The wound is then filled by a flap of cornea, taken in the same way from a human eye that has had to be enucleated, which is secured in place by sutures in the conjunctival flaps above and below. No stitches in the cornea are necessary. Nutritive conditions are favored by the fact that the flap lies upon a large wound surface and the conjunctival flaps, which quickly agglutinate, favor restoration of the circulation from the limbus.

Keratoplasty has long been a subject of interest to ophthalmologists. Many extensive opacities resulting from burns or ulcers are met with in eyes which are otherwise in good condition, but have their function destroyed by this mechanical impediment to the entrance of light. Ever since the beginning of the last century various methods have been tried repeatedly to replace opaque portions of the cornea, but they have been abandoned generally as fruitless. The repeated attempts that were made to replace the centre of the opaque cornea by a very complicated insertion of a piece of glass seem strange to us. Such a foreign body must soon be either cast off, or covered by granulation tissue, so that the optic effect is illusory. An attempt to transplant transparent cornea from an animal, or better from man, is more promising. A distinction is to be made between total and partial keratoplasty; in the former a portion involving the entire thickness of the cornea is excised by means of a trephine, in the other the upper, opaque layers alone are removed, leaving *Descemet's* membrane intact. Total keratoplasty is objectionable because the iris comes into apposition with the margins of the wound, the lens is endangered during the operation and infections can readily find their way into the eye. It is very rare that the flaps of human or animal cornea employed to fill the aperture remain transparent. *Zirm* performed a successful operation in this manner in one case. Even *v. Hippel*, who originally believed total keratoplasty to be the only operation from which anything could be hoped, recommended later that the operator confine himself to the removal of only the anterior layers, and replace them with clear corneal tissue taken either from man or an animal. Hitherto the results have not been satisfactory, although some cases have been reported in which the flaps adhered and were still transparent after the lapse of several months. As the possibility has been proven that a transplanted piece of cornea may remain transparent after a partial keratoplasty it seems to be probable that better results may be obtained from an improvement in the technique of the operation. Experiments, recently instituted at my clinic by *Loehlein*, indicate that faultless fixation of the flap in the wound and favorable conditions for its nutrition are essential for its success, and *Loehlein's* new operation complies with both of these requirements. Unfortunately so successful a keratoplasty as this one cannot ordinarily be attained, because a clear human cornea from an eye that needs to be enucleated is rarely at our disposal. It has not yet been determined whether material suitable for transplantation can be obtained from the cornea of a monkey.

A second procedure, which likewise can be carried out only in the clinic, may be illustrated by a number of trachoma patients. The infusion of the jequirity bean was introduced into ophthalmology by *de Wecker* for the treatment of vascular opacities of the cornea. The active agent in the infusion of jequirity is a toxic substance, abrin, which, when applied to the conjunctiva, produces a severe inflammation called jequirity ophthalmia. The result of this ophthalmia in many cases was a reduction of the superficial opacities in the cornea and a great improvement of the vision, but this form of treatment had to be abandoned because the degree of the inflammation produced could not be gauged when the infusion was used. The problem how to graduate this inflammation had to be solved in order to utilize the occasionally unmistakable advantages of jequirity. This I have done, using the first investigations of *Ehrlich* as a basis, by obtaining a constant solution of abrin, something which is not easily accomplished with toxins. Then I prepared definite dilutions of the toxin and finally produced a specific antitoxin through experiments on animals. This remedy is known as jequiritol. Instillation of some of the toxin into the conjunctival sac of either an animal, or of a man, is followed by a sharp inflammatory reaction of the conjunctiva and a serous infiltration of the cornea. I have been able to demonstrate that hand in hand with this inflammatory reaction the conjunctiva acquires a specific immunity which enables it to stand larger and larger doses of the toxin. At the same time the entire body gains an immunity to the poison in consequence of the development of specific antibodies, as has been shown by *Ehrlich*. Finally the strongest doses excite no reaction and then the remedy has lost its efficiency. On the basis of these facts it is easy now to induce a series of artificial inflammations of the eye by increasing doses of jequiritol until the stage desired for therapeutical purposes is attained.

In this first patient the jequiritol ophthalmia is at its height. Both eyelids are swollen, œdematous, reddened and hot, while the preauricular gland is somewhat swollen. A fluid, serous exudate wells out from the palpebral fissure. The conjunctiva bulbi is chemotic, the conjunctiva tarsi is covered with adherent, yellowish membranes. The picture resembles that of conjunctivitis crouposa. In the cornea the vessels of the pannus are distended with blood.

In the next patient the ophthalmia has subsided; the lids are no longer swollen, the membranes have been cast off, the conjunctival secretion has almost ceased, but the whole of the mucous membrane still is greatly relaxed and the conjunctiva bulbi remains much injected. The changes in the cornea are of greater interest. A comparison of the two eyes shows that the opacity is more dense in this one than in the eye that has not been treated and the patient states that his vision is worse than it was before. So it must be. We know that in a few days the cornea will begin to clear up. The vessels in the pannus are strongly injected and the movement of the blood through them can be seen with the aid of *Zeiss'* loupe. The entire cornea is infiltrated with serum and a new formation of vessels can be perceived. Gradually light places appear about the vessels, the iris becomes more distinctly visible and finally an involution of the vascularization takes place. The time required for this process varies a great deal; it depends on the thickness of the pannus, on the severity of the jequiritol ophthalmia, and on individual circumstances.

In the next case a comparison of the two eyes, which formerly were equally opaque, shows the full result of this treatment. In the untreated eye the pannus is unchanged, in the other the cornea is remarkably transparent. Vessels can still be seen, for they never disappear entirely after so grave a pannus. The patient says he is fortunate in being able to see better once more.

The use of this remedy is indicated in superficial, vascular pannus, especially when due to trachoma, after other methods of treatment have failed. The eye must be examined carefully before the instillations are made; the function of the lacrymal passages must be normal and there must be no ulcer on the cornea. Some drops of toxin No. I are instilled and by the next day we know whether the mucous membrane is particularly sensitive. In the great majority of patients this is not the case, and then on the second or third day No. III, or more often No. IV, is administered. The use of the strongest solution is advantageous because only a drop or two needs to be used and less escapes from the conjunctival sac. After the instillation the patient compresses the lacrymal sac and keeps his head inclined outward for some time in order to prevent as far as possible the passage of the poison into the nose. After an incubation period of a few hours the signs of ophthalmia commence. The conjunctiva and lids become swollen, secretion appears and membranes develop on the conjunctiva tarsi. The inflammation reaches its height in about twenty four hours and then slowly declines for a few days until it disappears. When jequiritol is used in this careful manner the antitoxin I produced experimentally is not needed. The cornea may be cleared up remarkably in selected cases and not a few of my trachoma patients who were scarcely able to see to go about have been enabled to return to work. It must be remembered that this is a toxin which requires exact indications and careful use. The increasing employment of it, especially in trachomatous regions, and the results obtained prove that I have placed on a rational basis the jequirity therapy which was once discarded on account of its danger, and have thus added a remedy useful in many cases to the therapeutic armamentarium of the ophthalmologist.

We come now to the remedies for corneal opacities which may be used by the general practitioner. The process of involution is so slow that these patients must receive ambulant treatment for a long time after their discharge from the clinic.

A remedy which is much used at present is dionin, introduced into ophthalmology by *Wolffberg*. An instillation of dionin into the conjunctival sac induces a marked hyperæmia and œdema of the conjunctiva and lids, associated with a sharp feeling of burning and lacrymation. The vessel walls are rendered more pervious by this serous infiltration, or lymphatic inundation. The reaction is useful in the clearing up of corneal opacities. Dionin also acts as an analgesic, as was established by *Darier*. The eye becomes accustomed to this remedy with remarkable rapidity. We instill weak solutions at first and later employ 5 or 10% solutions. After the remedy has been used for four or five days in succession no reaction can any longer be obtained. It is well to instill a drop of cocain beforehand in order to prevent the burning.

Subconjunctival injections of salt solution may be used in many cases, as in the regressive stage of a parenchymatous keratitis.

The principal remedy for the general practitioner to use in order to clear up the cornea is massage with yellow ointment. The eyes should be massaged for weeks and months in eczematous, or interstitial keratitis, while the strength of the ointment may be increased gradually from ½% to 4%. The ointment is placed in the conjunctival sac by means of a glass rod, and a piece of cotton is placed over the closed eye which is then rubbed with the fingers. We know little about how these remedies produce their effects. They all cause a transient hyperæmia of the eye and therefore exert some influence over the circulatory conditions. We use irritants to clear up corneal opacities, but with our present knowledge we cannot penetrate the mystery that enshrouds their effects upon the tissues and conceals so many problems.

40. Keratitis Dendritica and Herpes Corneae

This child, according to the statement of his physician, has been attacked by a peculiar disease of the cornea while he was convalescent from a severe febrile bronchitis. The cornea is surrounded by a marked ciliary injection and has in its centre an extremely graceful opacity. A narrow, gray cloud runs in zigzagging lines which give off little branches with nodular swellings, infiltrates, at their extremities. The lesion is superficial and the epithelium appears to be abraded over the whole of this arborescent figure. When stained with fluorescin it becomes evident that these lines are superficial furrows in the cornea with gray, infiltrated margins. This disease is known as keratitis dendritica. It belongs to the group of herpetic affections of the cornea and is usually met with after influenza, or other severe catarrhal diseases. The symptoms of irritation, photophobia and blepharospasm, are very severe. The treatment of this disease consists of atropin, heat, bandaging and sublimate ointment locally, together with aspirin internally. The disease is usually quite obstinate and is apt to leave behind it an opacity of characteristic shape.

This herpes corneæ febrilis frequently appears under other forms. Herpetic vesicles appear on the lips, nose, or ears after febrile diseases of the respiratory organs, disturbances of the digestive tract, or other infectious diseases, and, if the cornea is involved, a minute vesicle is formed, which is as clear as water and is accompanied by severe symptoms of irritation. This vesicle is a collection of clear fluid beneath the epithelium. When the eye is examined the vesicle has usually been ruptured and only its remains can be found in the form of little shreds of epithelium hanging from the cornea. Then the disease is known as filamentous keratitis. The clinical picture resembles that of a traumatic erosion, which was portrayed under injuries of the cornea. If an attempt is made to remove the diseased epithelium it will be found to be loosened over a greater area than would have been supposed a priori. Microscopical examination of such an epithelial filament reveals a vesiculation and swelling of the cells, as well as an intracellular œdema. Whether the ease with which the epithelium can be detached is to be ascribed to an œdema of the parenchyma of the cornea, or to a primary disease of the epithelium is uncertain, normally the epithelium is very adherent. Severe coryzas may be followed by a so called keratitis superficialis, which greatly resembles herpes corneæ. Numberless little spots appear over which the epithelium is clouded as if dusty, and may look as if inflated. The real cause of these forms of herpes is still unknown. The cornea is also involved in herpes zoster ophthalmicus.

41. Changes in the Form and Size of the Cornea

The changes of form most frequently met with in the cornea are *keratoconus*, *keratoglobus* and *staphyloma*.

Here is a patient in whom the change of form is the most prominent symptom. If we look at the profile of the eye, we can see that the cornea has the form of a truncated cone with its apex at, or a little below the centre. This condition is known as keratoconus.

If such an eye is examined with a keratoscope, the reflex image is seen to be greatly diminished in size at the apex of the cone and to be lengthened as soon as the eye moves, in consequence of the anomaly of curvature. If an examination is made with the skiascope, the most varied conditions of refraction are found to exist close together; in the centre is myopia, which quickly changes to hypermetropia.

After the eye has been cocainized, if a probe is pressed carefully on the apex of the cone the tissue is found to yield readily to the pressure, which proves that the cornea is very thin at that place. Keratoconus may be congenital, and then other anomalies are often found in the eye, but usually it appears at the age of puberty without having been preceded by any inflammation. It may affect either one eye, or both, and is more frequently met with in women. After it has lasted a considerable time the apex of the cone is apt to become opaque in consequence of a rupture of *Descemet's* membrane. When the cornea is very thin it may have a rhythmic, pulsating movement. As a rule the anomaly of curvature finally remains stationary, but the danger of rupture is very great. The cause of this disease is unknown; it has been ascribed to a slight power of resistance on the part of *Descemet's* membrane. The patients come to us because of their visual troubles and we have to try to fit them with suitable glasses. In some cases stenopaic slits are of advantage, in others cylindric, or hyperbolic glasses. As long as the centre of the cornea remains transparent we content ourselves with these means. In case of necessity we may cauterize carefully the apex of the cone, in order to flatten the curvature by cicatrization, and then perform an optic iridectomy. In other cases the hydrodiaphanoscope of *Lohnstein* may be tried. This is a cup which, when filled with a proper salt solution, is applied close to the margin of the orbit; over this a correcting lens is added.

Another change in the form of the cornea is keratoglobus. This is met with congenitally in abnormal enlargements of the eye, such as hydrophthalmos. The changes in form met with most frequently in practice are the staphylomata, which have already been studied as sequelæ of corneal ulcers.

42. Keratitis Disciformis

A few weeks ago, in all probability, this patient had a slight injury of his eye, after which a peculiar, gray, disc shaped opacity developed in his cornea with comparatively slight signs of irritation.

It is difficult to determine whether this is a disease of the superficial, or of the deep layers of the cornea; the infiltration certainly extends from the surface deeply into the tissues. In the centre of the disc is an intensely opaque place which evidently is the principal focus of the inflammation. From this place the cloudiness grows thinner toward the periphery, and at first the marginal part of the cornea remains clear. In a later stage both deep and superficial vessels may extend from the margin of the cornea into the area of the disc. If we examine the disc with the aid of *Zeiss*' loupe radiating branches can be seen to extend in all directions from the dense central infiltrate, which in turn are covered by delicate clouds. The appearance is suggestive of a colony of some fungus lying in the parenchyma of the cornea and sending out ramifications.

A third characteristic of the disease is that the epithelium is dotted over all the diseased portion of the cornea. In the centre of the disc, over the dense infiltrate, a slight defect in the epithelium is often to be found, but it almost never goes on to ulceration. In this respect the disease bears a certain resemblance to interstitial keratitis.

A fourth typical feature is that the sensibility of the cornea is markedly reduced over the diseased portion, while it remains normal elsewhere.

A fifth point is the extremely slow course of the disease. Months frequently elapse before the opacity changes its character. Although such an opacity does clear up more or less, especially in the periphery where it is thin, quite dense opacities are left. From this fact the prognosis may be inferred. Relapses are not infrequent, sometimes after an interval of months, and the patient may return with even a hypopyon in the anterior chamber. The picture as a whole gives the impression that a focus of infection is situated in the cornea, which is not virulent, but against which the cornea is unable to defend itself. A similar clinical picture has repeatedly developed after a vaccine infection of the cornea. The cause of keratitis disciformis is still unknown. The treatment is purely symptomatic; constant heat to the eye, bandage and atropin in the early stage. Subconjunctival injections of salt solution may also be used. In the later stages as great an involution and clearing up of the opacity as possible is sought to be obtained by massage. Probably it is caused by an ectogenous infection in connection with the injury.

43. Degenerative Conditions of the Cornea

Although the clinical picture about to be briefly demonstrated is of no great practical importance it is one with which all should be acquainted, because it shows that grave degenerative processes may take place in the cornea. The primary trouble in our patient is a phthisis bulbi, the result of an injury many years ago. A white, or partly brownish opacity extends from the outer to the inner margin horizontally over the middle of the cornea, which is rather small, in the region of the palpebral fissure. This is the ribbon shaped, or zonular opacity of the cornea. It can be seen on close examination that the principal changes lie in the middle of the opacity where large and small white, or yellow spots lie separated by transparent spaces.

Microscopically we find beneath the epithelium a vascular connective tissue which has partially broken up and destroyed *Bowman's* membrane. This connective tissue contains calciferous or homogeneous concrements of various shapes and sizes.

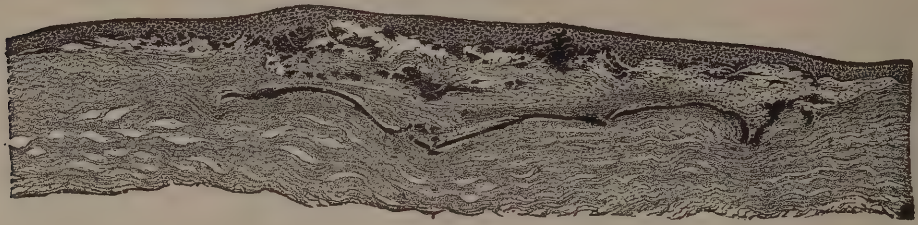
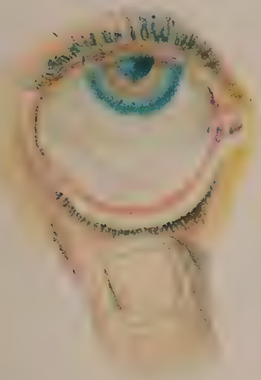
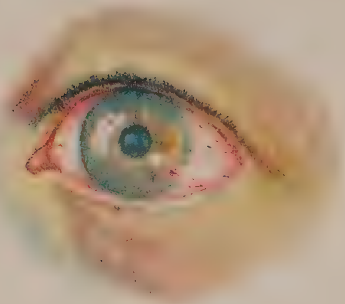


FIG. 30.—Ribbon Shaped Opacity of the Cornea.

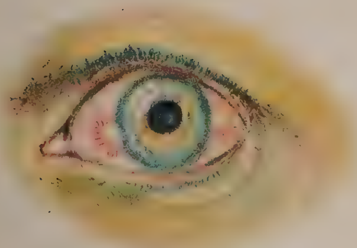
The chalky concrements especially are intensively stained. Evidently the nutrition in such an eye is greatly impaired. The portion of the cornea that lies in the palpebral fissure is the part most exposed to slight mechanical injuries, so it is in this region that the cornea reacts first with the formation of a vascular connective tissue in which, according to *Best*, secondary degenerative processes take place and result in a coagulation necrosis and in the formation of calciferous and hyalin products. I agree with *Vossius* that the calcification cannot be considered primary, as many have believed it to be. We meet with this and analogous degenerative processes most frequently in the corneæ of blind, atrophic eyes, sometimes in eyes with absolute glaucoma. Frequently these atrophic eyes contain scales of bone and calcareous deposits. It is worthy of notice that a brown discoloration of the cornea may be produced in the region of the palpebral fissure with aniline colors. A ribbon shaped opacity in the cornea of a phthisical eye always indicates that the nutrition of the eye is seriously impaired. Attempts to scrape out such masses should be made only when particularly desired by the patient, enucleation should be recommended otherwise. Cases are met with in which similar changes have taken place in an eye which still retains useful vision; in these the attempt to remove the masses should be made, because they are apt to excite severe irritation.



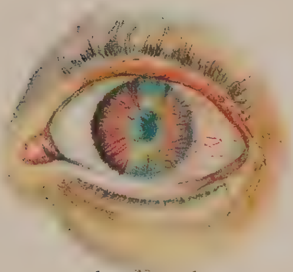
a Symblepharon.



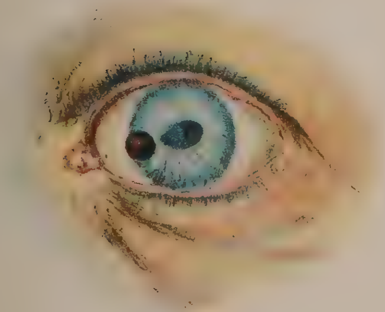
b Ulcus eczematosum migrans.
(Vascularized strips tissue.)



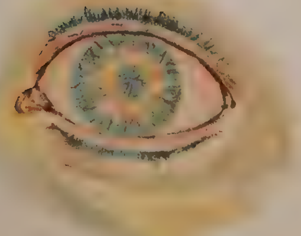
c Keratoconjunctivitis eczematosa.



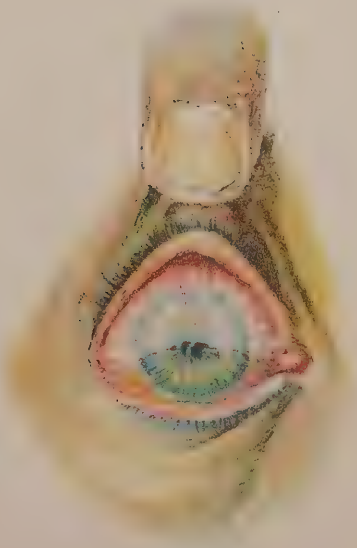
d Development of vascular channels in the cornea (schematic).



e Prolapse of the iris, sequent to the perforation of an eczematous ulcer.



f Pannus eczematosus.



g Pannus trachomatosus.

III

Diseases of the Iris

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44. Rheumatic Iritis and the Fundamental Principles of the Treatment of Iritis

We have already studied the most essential symptoms met with in the so called primary iritis, ciliary injection, contraction of the pupil, discoloration, indistinctness of details and formation of exudates, so the student recognizes the condition in this case and denominates it correctly a fibrinous iritis.

I take this opportunity to reiterate the fact that in certain diseases of the eye the ophthalmologist should not devote his attention wholly to that organ. The appearance of an iritis is a monition to the general practitioner to turn his attention to other parts of the body also, for the fundamental proposition is inculcated that every primary iritis is but the local expression of another, general disease. In the examination of a case of iritis the question instinctively and immediately arises: How is it possible that an inflammation of the iris can develop, either suddenly or gradually, in a healthy eye? As soon as the diagnosis is made the etiology must be determined, for the treatment should be directed toward the cause. Almost every grave general disease may cause an iritis, but those which have chiefly to be considered are rheumatism, gonorrhœa, gout, diabetes, syphilis and tuberculosis. Therefore the general practitioner as soon as he sees an iritis will make a general examination and govern his treatment according to his findings.

The special duty of the ophthalmologist is different; he should try to ascertain the etiology of the iritis in an individual case from a careful study of the changes in the eye. This cannot always be done; an examination of the whole body is necessary in most cases and such an examination is made in every case in our clinic. The result of the general examination of this patient indicates chronic articular rheumatism as the cause of his iritis.

Still a careful analysis of the changes in the eye may facilitate the recognition of the etiology.

All forms of iritis may be divided into two large groups: those in which the anterior surface of the iris is the part chiefly affected, and those in which the inflammation involves principally the deeper layers. An iritis in which the anterior surface is the part of the iris chiefly affected is probably an iritis rheumatica, an iritis gonorrhoica, or an iritis urica.

Although the cardinal symptoms of an iritis are always the same and the cases appear very similar, the limitation of the inflammation to the anterior surface of the iris may be detected by a careful examination with *Zeiss'* loupe.

The eye has a very marked pericorneal injection. In the iris, the details of which are rather indistinct, single hyperæmic vessels can be seen to radiate from the periphery toward the pupil and to extend in part over the sphincter to its margin. Two other symptoms are quite marked, one on the anterior surface of the iris, the other on the edge of the pupil.

Little deposits, from some of which fine threads extend, are scattered in various places over the anterior surface of the iris. They are of a delicate gray color, consist of exudates of fibrin and, together with the hyperæmia, cause the indistinctness of the details. The iris itself seems to be only slightly swollen.

I call particular attention to the linear, seamlike agglutination of the lower margin of the pupil to the anterior capsule of the lens. The brown edge of pigment alone is principally involved in this agglutination. The rest of the pupil is free and has been retracted uniformly by the atropin. Deposits may also be perceived on the posterior surface of the cornea.

The course of a rheumatic iritis is often typical. At first the patients complain of lacrymation and photophobia and show no symptoms of iritis. There is rather a hyperæmia of the conjunctiva, sometimes a distinct conjunctivitis with a small amount of secretion, so that the correct diagnosis is not made. After a few days the hyperæmia of single superficial vessels in the iris can be recognized together with a ciliary injection. At this time the pupil may exhibit its first tendency to contract, in other cases there may be a slight mydriasis. As the inflammation proceeds scattered deposits of fibrin appear on the anterior surface of the iris, which may not always be separate, but the entire iris and pupil may be covered transiently by a layer of fibrin, which may be stripped off by dilatation of the pupil with atropin.

In this stage occur the adhesions at the margin of the pupil which involve really the brown pigment edge. There is no swelling of the tissue in the pupillary portion of the iris and there are no circumscribed foci in the deeper layers at the margin of the pupil, which we will learn to recognize in other forms of iritis and are productive of broader synechiæ.

These pigment layer synechiæ exhibit a striking predilection for the lower part of the pupillary margin in rheumatic iritis. According to *Krueckmann* the synechia does not need to be formed where the fibrin is excreted, but the fibrin may sink downward, in consequence of the upright position of the head, and adhere to the lower margin of the pupil. When patients lie for a long time on one side the principal synechiæ may form on either the temporal, or the nasal side of the pupil.

That these posterior synechiæ formed in a rheumatic iritis are in fact agglutinations of the pigment layer without involvement of the connective tissue of the iris is shown first by their linear and seamlike nature. This is brought out clearly by the effect of atropin. The synechiæ may be ruptured and then it can be seen that only parts of the retinal pigment layer remain attached to the lens capsule. When the synechiæ are not ruptured, and the iris is only retracted somewhat, the elongated digitations can be seen to consist almost entirely of pigment epithelium. The places where the pupil dilates, when such synechiæ are present, have the shape of larger or smaller segments of circles, they are never pointed, or angular, as in other forms of iritis.

When the patient has a blue, or a gray iris the deposits on the posterior surface of the cornea contain no pigment, at least until the inflammation has lasted a considerable length of time, but when the iris is brown they are pigmented. The deposits are usually small and confined to the lower part of the cornea, but the longer the inflammation lasts the worse they become and the more irregularly are they distributed.

Finally a peculiar symptom frequently met with in rheumatic iritis can be seen with the loupe. This is a peculiar lattice work on the posterior surface of the cornea, which is conceived to be a wrinkling, or reticulation of *Descemet's* membrane.

As the ciliary body is apt to become involved in iritis the sensitiveness of the eye to pressure should be tested. In most cases of acute rheumatic iritis the ciliary body is involved comparatively little. In grave cases and recurrences the appearance of opacities in the vitreous is an evidence that the inflammation has extended to the ciliary body.

Reviewing our examination of this case we have found two distinctive features: the disseminated deposits of fibrin on the anterior surface of the iris, and the seamlike agglutination of the retinal pigment layer at the margin of the pupil. What deduction can be drawn from these concerning the etiology of the disease?

The ophthalmologist can conclude that this is a superficial inflammation of the iris. Thus far our clinical diagnosis extends, but no farther. Even this diagnosis could not be reached, if we were not able to study these minutiæ of the changes in the iris by means of *Zeiss*' loupe. We have certainly learned something, for investigations have shown that the preeminent causes of superficial inflammations of the iris are rheumatism, gonorrhœa and gout. Thus the careful analysis of the changes present has a certain importance in the differential diagnosis. We learn thus to distinguish superficial from deep inflammations, but in many cases the lines which separate them are blotted out. The etiology in the individual case cannot be determined from the superficial inflammation. This forms only an indication, the diagnosis must be made through a general examination.

The question arises: How far can a practicing physician analyze such an iritis? He can scarcely have at his disposal so costly an instrument as *Zeiss*' loupe and the simpler binocular loupe is often insufficient. By means of a single magnifying lens in combination with focal illumination the posterior synechiæ at least can be seen considerably enlarged and their nature determined. But the general diagnosis of iritis is sufficient; its etiology can be determined better from the general examination than from a detailed analysis of the local changes in the iris.

The etiology of acute and chronic articular rheumatism stands in need of further investigation, but there is scarcely any doubt that in rheumatism iritis is produced by metastasis. It has been shown that the anterior layer of the stroma of the iris, in which the inflammation takes place, forms a wall to a space that contains fluid in a manner analogous to the synovial cavities of the joints.

It is quite probable that the agent of rheumatic iritis is not very pathogenic, because no pronounced injury of the tissue persists in the places where the excretion of fibrin takes place on the anterior surface of the iris, and particularly because the inflammation is not of a purulent character.

Slight attacks of rheumatic iritis may pass away in a few days, but usually recovery requires a longer time, it may be several weeks. Relapses are very apt to occur with fresh colds.

Treatment of rheumatic iritis. It is self evident that the treatment of every case of rheumatic iritis, or iridocyclitis, must be both general and local. In acute iritis the patients belong in bed. The diet should be regulated and adapted to the etiology of the disease. All excitants that tend to raise the blood pressure are done away with. The general treatment is that ordinarily employed in articular rheumatism. When the joint disease is fresh we prescribe internally preparations of salicylic acid, particularly aspirin and diplosal, and externally local embrocations with *Burow's* solution, alcohol, or mesotan. In chronic rheumatism baths are of the first importance. In the clinic and at home we use simple warm water baths of a temperature of 30° to 35° C. for 15 or 20 minutes two or three times a week, keeping the patient in bed during the intervals if he has iritis. If the joints exhibit fresh exacerbations of rheumatism warm packing and embrocations are indicated. The internal treatment of chronic rheumatism includes the administration of potassic iodide, wine of colchicum, arsenic, the preparations of lithia and the alkaline mineral waters. The patient must obtain sufficient rest at night and when necessary this must be secured by means of narcotics. The pains that accompany iritis, and radiate into the brow and temple in the region supplied by the first branch of the trigeminus, are sometimes agonizing. They are spoken of as ciliary neurosis. After the iritis has run its course the patient is transferred to his physician for further treatment of his rheumatism. Thus the general treatment varies according to the etiology of the iritis, the severity of the disease and the personality of the patient, but the local treatment of the eye is the same in all forms. I am convinced that an iritis can be treated best and most safely in a hospital, but this is not always feasible.

The local treatment of every case of iritis begins with the regular instillation of a 1 or 2% solution of atropin. Instead of solutions we may use atropin ointment, which is very suitable and more constant in its effect, because it is not washed away so soon by the tears. The atropin is administered from two to four times a day according to the severity of the case.

Atropin is not tolerated uniformly. In many persons it excites an irritation of the conjunctiva and even an œdema of the lids after it has been used a few times. In other persons a peculiar follicular inflammation of the conjunctiva, which presents a certain resemblance to trachoma granules, appears after the drug has been employed for some time. It should also be remembered that atropin may excite toxic symptoms. The first complaint of these patients is of dryness in the throat and difficulty in swallowing. In grave cases of poisoning the salivary and perspiratory secretions cease (paralyses of secretion). Ephemeral erythemata appear on the skin, the heart's action is disturbed and conditions of nervous excitement, even accompanied by hallucinations, appear, especially in old people. A subcutaneous injection of morphin is indicated as an antidote. In all such cases scopolamin is employed as a substitute for atropin. A ¼% solution of scopolamin is less toxic than a ½% solution of atropin.

Atropin can increase the intraocular tension, so it is necessary to use a meiotic in many cases of cyclitis in which the tension is increased, but nevertheless atropin remains the principal remedy in iritis.

Atropin is employed for two reasons: First, to keep the inflamed tissues as quiet as possible. Atropin paralyzes the sphincter, dilates the pupil and prevents its to and fro movement. The iris becomes narrower and less in volume because lymph is squeezed out of it and the influx of blood is lessened. In this way atropin acts directly upon the inflammation.

It is used also to break up existing synechiæ when possible and to prevent their formation with a contracted pupil. Even the formation of connective tissue synechiæ may be restrained by the mydriasis in this way. The granulation tissue, which takes the place of the fibrinous agglutinations, comes chiefly from vessels which sprout out of the capillary area of the sphincter, but when the sphincteric portion of the iris is drawn toward the periphery the formation of these vascular sprouts, and therefore the development of connective tissue synechiæ, is rendered difficult. Even when some do form while the pupil is dilated, they occupy but a small part of the pupillary circle, while with a contracted pupil they may readily occupy the whole circumference. Thus atropin prevents seclusio pupillæ.

Atropin likewise paralyzes the ciliary muscle and so keeps the very vascular near neighbor of the iris quiet.

In very grave cases the effect of atropin may be enhanced by the addition of cocain, which stimulates the dilatator and so increases the mydriasis.

After the atropin has been administered the eye is covered with a moist dressing and the application of heat to the eye is begun immediately. When once you have heard a patient with iritis exclaim how grateful the heat feels to the eye, you will never think again of trying to combat the inflammation with cold. Eyes with iritis have a great need of heat, which may be applied in many ways. Many old methods are not to be despised. Even in the poorest hovel the mother can prepare hot poultices. Cherry stones and plum stones gathered, dried, sewed into little bags and heated in the oven may do good service when laid upon the eye.

Here in the clinic we use in all such cases an electric thermophor with which we can gauge the degree of heat. Finally a word of advice. The reaction of the pupil to atropin gives the best indication regarding the course of the disease. If the pupil remains contracted the patient should be transferred to the clinic, because of the danger of secondary glaucoma.

45. Iritis After Gonorrhoea and the Operative Treatment in Chronic Iritis

The presence of the cardinal symptoms, ciliary injection, discoloration of the iris, indistinctness of its details, formation of exudates, indicate this to be a case of iritis. As there are no signs of a discrete inflammation and as the synechiæ are seamlike, it appears to be a superficial inflammation. Our inquiries concerning its etiology are facilitated by the admission of the patient that he has a gonorrhœa.

Iritis occurs very rarely during the acute stage of a urethral gonorrhœa, but in the late stages of that disease it is very common, especially in association with gonorrhœal rheumatism. We have to consider gonorrhœal iritis as a gonococcal metastasis. We know that gonococci usually are only local agents of inflammation in certain mucous membranes, and it coincides with our knowledge of bacteriology that the infections produced by these agents, when they enter the circulation from these foci and are carried to the iris, are exceptionally virulent. The metastatic gonorrhœal inflammations and suppurations of joints are very serious diseases. Of course the patients have a certain susceptibility, a reduced power of resistance to the occurrence of these metastases, but in order to understand the clinical picture of gonorrhœal iritis it must be known that even the most virulent gonococci cannot live long in the blood.

Let us return to our patient. In the first place this is not a purulent iritis, fibrinous exudates only can be seen at the margin of the pupil. At the commencement of a gonorrhœal iritis a little exudate, which resembles hypopyon, is found not infrequently at the bottom of the anterior chamber, but it consists principally of coagulated fibrin and soon disappears. Many ophthalmologists are of the opinion that this exudate at the onset of an acute, superficial iritis is diagnostic, but I cannot agree with them. The kind of exudate at the margin of the pupil is more characteristic. Usually quite seamlike, linear agglutinations of the margin of the pupil to the anterior surface of the lens are present. The brown agglutinations and the brown deposits have distinctly the form of concentric rings, especially after several recurrences, and the margin of the pupil is not notched as in forms of iritis with more discrete inflammations. But these synechiæ are not so characteristic that a diagnosis of gonococcal iritis can be based upon them. Other distinctive features must be looked for.

It has been pointed out that the onset of a gonorrhœal iritis differs from that of a rheumatic, in that as a rule it is acute and associated with contraction of the pupil, but this distinction is by no means reliable. We can only say that gonorrhœal iritis is a superficial, nonpurulent disease of the iris. No tissue necrosis occurs and only small deposits of fibrin in the iris can be detected with the loupe, but these symptoms are shared by the other superficial inflammations.

As a rule gonorrhœal iritis occurs only when the joints have been attacked, and may appear suddenly after the urethral inflammation is well. In this form of iritis also the diagnosis often cannot be made until after the general examination.

It has already been mentioned that in these cases it is evident that virulent gonococci have entered the circulation. Gonorrhœal iritis is sufficiently grave, even if these agents do not cause sepsis, for clinical experience has demonstrated it to be one of the most protracted of the superficial inflammations of the iris. It is not known how quickly, or how slowly the gonococci die in these metastases in the iris, but it is plain that after their death endotoxic substances continue for a long time to excite inflammation.

Relapses are very frequent, often one attack follows another. At first the iris alone is involved, but later the inflammation extends to the ciliary body; then fine dustlike opacities appear in the vitreous and impair the vision. Pain also is not lacking, as this patient can testify. The chorioid is remarkably exempt from these gonococcal metastases. A chorioretinitis in association with gonorrhœa has been described in only a few cases. The patients are almost always men, women are affected much more rarely.

As a rule both eyes are affected, though it may be in turn and after an interval. I have an idea that the distribution of the vessels in the iris is not the sole determining factor in the localization of the gonococci, but that specific affinities exist between them and the cells.

The local treatment of the eye is just the same as in other forms of iritis, mydriatics and the application of heat. It need only be mentioned that the fundamental disease, the urethral gonorrhœa, must be treated in a proper manner at the same time.

I have another patient on whom I can demonstrate the operative treatment of iritis. The history is that the man has had a number of recurrent attacks of iritis, which was due in this case also to gonorrhœa. Operative intervention is frequently necessary in such an eye as this, whatever may have been the etiology of the iritis.

I advise that hospital treatment be insisted on in any case of iritis in which the pupil reacts badly, or not at all to atropin, because in such a case seclusio pupillæ is either present, or to be feared. Such a condition is present in this case. The eye is pale, the pupil is very small and irregular, while the whole of its margin is attached to the anterior capsule of the lens by brown and gray tissue. Atropin produces no effect upon it and the intraocular tension is decidedly increased. Although there is no recent inflammation the iris has lost its beautiful appearance. Some folds look like cords, as they are no longer enveloped as formerly in loose tissue. This is the picture of a well marked atrophy of the iris. In such an atrophic condition the iris may become so thin that the black pigment epithelium may show through.

Atrophy of the iris occurs in many conditions. In old age the iris may become somewhat atrophic in consequence of arteriosclerosis, but the principal cause of atrophy is a stretching of the membrane. In this case the seclusion of the pupil has caused the membrane to become stretched. The same thing can be seen in a leucoma adhærens. Partial atrophies in the region of the sphincter are also met with in association with discrete synechiæ.

The treatment for such a condition as this is iridectomy. The indications for this operation are self evident when symptoms of secondary glaucoma have appeared, but it is better not to await the appearance of such symptoms with their attendant dangers. I frequently perform iridectomy for iritis after one or more attacks of inflammation when recurrences are to be expected. According to the experience of both myself and others iridectomy in chronic iritis is an efficient means to prevent recurrences. At least we have then done all in our power to guard against a seclusio pupillæ.

The iridectomy is made upward. The eye should be free from recent inflammation at the time of the operation, because otherwise the coloboma readily may become closed again by a large exudation. The bleeding after an iridectomy in this condition is always greater than usual, because of the hyperæmic state of the tissues. Even when no fresh inflammation is perceptible clinically such tissues react readily with large hemorrhages.

Sometimes, when the synechiæ are not too extensive and do not contain very much connective tissue, we may avail ourselves of this opportunity to divide them by means of a small spatula, but this manœuvre must be performed with extreme care because the capsule of the lens is easily torn. The after treatment of the operation is the same as usual, rest in bed and a bandage. As a rule the absorption of the hyphæma takes a longer time than usual.

46. Iritis in Gout, Diabetes and Other Diseases

This is an elderly patient who had during the night a sudden attack of severe pain in the metatarsophalangeal joint of his great toe, followed by similar attacks for several nights. He has had such attacks before and knows that he has gout. But, while on previous occasions the attacks passed away without causing any other disturbance, this time he was suddenly seized with severe pain in both eyes in the middle of the night, simultaneously with the pain in the joint.

In each eye the whole of the conjunctiva scleræ is so extremely injected that we are obliged to think involuntarily of a commencing conjunctivitis, but closer inspection reveals a ciliary injection, deposits on *Descemet's* membrane and a moderate degree of discoloration and obscuration of the iris, symptoms indicative of a serofibrinous iritis. The changes in the toe joint are those characteristic of an attack of gout. This is not the place to discuss the great subject of gout, but we will not err when we connect the iritis with this disease. In fact this kind of iritis has been known for a long time.

This form of iritis also is superficial. Just as in the joint the surface, the cartilage, is first affected, so in the iris the richly cellular, but very anæmic anterior layer of the stroma is first attacked. The other layers of the iris and the ciliary body are involved only in very grave cases.

The iritis of gout often attacks both eyes, is very prone to recur, and is one of the most protracted forms of this inflammation. A striking symptom is the very great injection of the conjunctiva scleræ, in which an episcleral hyperæmia takes part, while the changes in the iris are comparatively slight. Gout is always to be thought of when the first attack of iritis occurs in a patient of advanced age.

The onset of the iritis is sudden, takes place at night and is accompanied by severe pain, exactly like that of the attack in the great toe. The parallelism is so characteristic that the paroxysms have been spoken of as intraocular attacks of gout. The iritis may also occur as a forerunner of an attack, as has been demonstrated by *Krueckmann.* The pain is more severe than in other forms of iritis, frequently it is extreme. Another feature of the clinical picture is that iritis urica occurs only with arthritic, not with primary renal gout. Finally it should be mentioned that its onset is not infrequently mistaken for an attack of glaucoma until the iritis becomes evident. The true nature of iritis urica is as unknown to us as the ultimate cause of the abnormality of metabolism called gout. We find no concrements, or deposits in the iris clinically.

We prescribe in this form of iritis the same therapy that is employed in the treatment of gout. When the joint is acutely inflamed it is wrapped up warmly, and sodium salicylate, aspirin, salipyrin, colchicum and potassic iodide are given internally. Simple milk diet and weak mineral waters complete the internal treatment. In the chronic uric acid diathesis the diet must be regulated carefully. But these are matters to be dealt with in the study of internal medicine. The local treatment of the iritis is the same as that of other forms, but as the pain is extreme narcotics are indispensable.

Iritis is met with also in other diseases of metabolism. Here is an elderly patient with a primary, purulent iritis. It is distinctly a fibrinous inflammation with a little hypopyon at the bottom of the anterior chamber. The patient exhibits no symptoms of rheumatism, gonorrhœa, syphilis, or tuberculosis, but an examination of his urine reveals a considerable quantity of sugar. The urine of every patient with iritis should be examined for the sake of certainty. This man did not know that he was diabetic.

The purulent nature shown by the hypopyon is frequently characteristic of the iritis of diabetes and sometimes the entire pupillary region is filled with a fibrinous membrane. Yet the course is usually very favorable, the exudates appear to be absorbed quickly. The occurrence of a primary iritis in diabetes is rare. Cases of iritis without hypopyon and with synechiæ have been met with associated with diabetes. Finally an iritis has occasionally been observed in many infectious diseases, such as pneumonia, typhoid fever, angina, influenza, etc.

47. Iritis Syphilitica Fibrinosa

We begin today the study of syphilitic diseases of the iris. I have said repeatedly that when an iritis is seen the etiological factors should be enumerated: rheumatism, gonorrhœa and gout on the one side, syphilis and tuberculosis on the other. The former represent the superficial diseases of the iris, the latter the diseases of its deeper layers.

The study of the etiology of syphilis has been greatly promoted by the discovery of *Schaudinn*, one of Germany's ablest investigators, whom a tragic fate has snatched away too soon, that the spirochæta pallida is its agent. In a syphilitic iris the spirochætæ must have entered by way of the circulation. As these agents, at least in adults, inundate the blood when they have passed the lymphatic glands it is evident that the first period of the specific infection is past when the onset of a syphilitic iritis takes place. Iritis syphilitica does not belong to the primary stage of syphilis.

The early exanthem of syphilis, the *roseola syphilitica*, has been seen in the iris in the sixth week after the first sign of the primary lesion, under certain circumstances still earlier, before the first sign of the cutaneous eruption. Unfortunately I am unable to exhibit this to you, because such cases hardly come to the ophthalmologist, the early roseola in the iris causes no trouble. It retrogrades quickly without mercury and is considered to be due to capillary emboli of spirochætæ.

Clinically, branching and radiating injections of vessels are seen on the surface, which differ in form and appearance from macula and roseola, but this difference is due only to the peculiar kind of capillary development and formation in the iris. The central part of the iris is provided abundantly with minute vessels and it is here that roseola of the iris is chiefly to be met with. In such cases minute, bright red, striated, or stellate superficial hyperæmias are to be seen, which originate from a bloodvessel that follows a circular course. Such localized hyperæmias are not to be found in the region of the sphincter, although here the capillary network is much more compact, as they are covered by the tissue. The early roseola of the iris is to be looked for clinically only in the anterior layer of the stroma, although this is the most anæmic portion of the iris. In the later stages of syphilis, transient roseola of the iris hardly occurs; almost all roseolæ that appear in the first and second year of the disease develop, without treatment, into papules. When this does not happen the roseola may properly be termed *recurrent roseola.* Both the physician and the ophthalmologist meet much more frequently with the symptoms presented by our patient, to whom we will now turn.

He complains of severe pains which radiate from the eye over one side of the head. The cardinal symptoms, ciliary injection, discoloration, blurred details and posterior synechiæ, are the same as in the cases of iritis already demonstrated and the diagnosis *fibrinous iritis* is correct, but two additional symptoms are presented; the entire iris is swollen and œdematous, and the posterior synechiæ have a peculiar indented form. Hemorrhages and gelatinous exudates into the anterior chamber are not uncommon, as well as deposits on *Descemet's* membrane.

We should never be contented with the examination of the iris alone in such a patient. The presence of deposits on *Descemet's* membrane indicates that the ciliary body is involved, and that the iritis is really an iridocyclitis. If the eye is examined systematically dustlike opacities may be found in the vitreous which indicate an involvement of the chorioid. A hyperæmia of the papilla is frequently met with which may increase to a true neuritis, associated with impairment of the vision and scotomata. The retina may be primarily diseased, or it may become involved secondarily to the iritis. The presence of deep infiltrations and opacities in the periphery of the cornea sometimes are suggestive of parenchymatous keratitis, but as a rule the differentiation can be made from the fact that in this disease the opacity occupies only a sector of the periphery. Our patient presents none of these symptoms, but I call attention to them to emphasize the point that the entire eye should be examined and watched in every case of syphilitic iritis. The clinical picture here is that of a grave, plastic iritis.

It has been shown that it is rather difficult to determine the etiology with certainty from the clinical appearance in the superficial diseases of the iris, iritis rheumatica, gonorrhoica and urica, and the same is true in iritis luetica fibrinosa. It will be admitted readily that this is not merely a superficial inflammation; the entire iris appears swollen and œdematous. It also exhibits a *discrete* character in spite of its diffuse appearance. Atropin has been administered freely and distinctly indented posterior synechiæ may be seen at the margin of the pupil.

The connective tissue layers of the iris take part in the formation of these agglutinations with the lens capsule, in contrast with the superficial inflammations in which the adhesions are confined mainly to the posterior layer of epithelium.

This observation proves that in spite of the diffuse appearance some little localized foci of infiltration must be present at the pupillary margin to give rise to these indentations.

The involvement of the deep layers of the iris, especially the swelling of the tissue of the sphincter, which is absent in the rheumatic and gonorrhœal forms of iritis, completes the picture. Sometimes the distention produced by the œdema is unevenly distributed. If the iris is pigmented the small foci of pigment become quite prominent in the general swelling of the iris and must not be mistaken for nodules.

A diagnostic characteristic sometimes met with is that deposits are to be seen in the quadrant of the cornea that corresponds with the most swollen portion of the iris, and that such deposits formed in a syphilitic iritis result in an infiltration of the adjoining posterior layers of the cornea, which remind the observer of an interstitial keratitis. *Krueckmann* has shown that these infiltrations leave marked cicatricial opacities in the cornea. Perhaps spirochætæ are carried by the deposits to these places where they enter the posterior lamellæ of the cornea.

All of these factors are absolutely insufficient to demonstrate the syphilitic etiology of a fibrinous iritis in very many cases. Every ophthalmologist must admit that frequently it is impossible to differentiate this from the gonorrhœal and rheumatic forms of iritis, particularly when the eyes have undergone several attacks. It is also evident that a fibrinous iritis which appears many years after a syphilitic infection may have been caused by something else.

Syphilis is assumed to be the cause when the fibrinous iritis occurs in a certain period of the disease. It does not appear ordinarily before the third month after the initial lesion and often is accompanied by exanthemata on the skin and mucous membranes, the same as angina specifica. The œdema and fibrin in a syphilitic fibrinous iritis is the more abundant the earlier the inflammation appears after the primary lesion. It forms the first symptom of secondary syphilis only in rare cases. In general it may be said that plastic iritis is accustomed to appear in the course of the first or second year after the infection, usually between the sixth and the twelfth months.

It has been claimed repeatedly that injuries of the eye favor an outbreak of iritis.

Statistics have shown that iritis occurs in at least 3 or 4% of syphilitics; probably the percentage is greater than this. Of the cases of iritis observed in eye clinics syphilis is the cause in about 25%. The disease is unilateral as a rule, but in about a fourth of the cases the second eye becomes diseased, usually after a longer or shorter interval. The affection is bilateral in only about 10% of the cases of iritis due to other causes. The therapy will be considered in connection with the other forms of syphilitic disease of the iris.

48. The Papular Form of Syphilitic Iritis

In the form of syphilitic iritis just studied the etiological diagnosis could be determined only from a general examination of the body, but this is not the case with the patient we are now about to examine.

The eye presents a great conjunctival and ciliary injection, with a moderate discoloration and obscuration of the iris, but the symptom which immediately attracts attention is the presence at the margin of the pupil of two nodes about as large as the head of a pin, of a reddish yellow, or gray yellow color. The color varies in different cases from bright red, gray red and brown red to gray yellow and yellow according to the proportionate relations between the blood and the cellular elements of the nodules.

By focal illumination hyperæmic little vessels can be seen on the top of the nodule and in its immediate neighborhood, while the nodule itself projects in the form of a hemisphere into the area of the pupil and into the anterior chamber.

These nodules at the margin of the pupil suggest syphilis at once to the physician. Their preferred and principal seat in the iris is the vascular region of the sphincter and its immediate neighborhood. When the patient acknowledges that he has syphilis no doubt remains that the nodule is a syphilitic papule. Iritis papulosa, like the specific fibrinous iritis, is met with in the secondary stage of syphilis.

These two forms of iritis belong together. Anatomical studies have shown that even in the fibrinous iritis little discrete infiltrations can be demonstrated with the microscope, which are not perceptible to the naked eye. In the second place posterior synechiæ frequently are seen to develop at the margin of the pupil, from which can be drawn the positive conclusion, which has been confirmed by anatomical examination, that circumscribed minute papules must have been present. There is therefore no sharp line of demarcation between papular and fibrinous specific iritis. We speak of papular iritis only when the formation of nodules is a prominent feature of the clinical picture.

Papular iritis is often called iritis gummosa. This is because papules of the iris originally were known anatomically as gummata. It is not always possible to determine, even with the microscope, whether the lesion present in the iris is a papule, or a gumma. In addition to this true gummata may appear in the iris during the secondary stage of malignant syphilis. Yet I believe, with many other ophthalmologists, that papular can be differentiated from the gummatous iritis more sharply than from the fibrinous.

The overwhelming majority of the cases of iritis occur during the secondary stage of syphilis; the gumma of the iris is therefore much more rare than the papule. A gumma is usually a solitary tumor which attains a considerable size and appears almost without exception at the ciliary margin of the iris, or in the ciliary body. It may undergo fatty or caseous degeneration. After recovery an extensive, white gray scar is left in the iris, which is adherent to the lens capsule. The gumma may also proliferate, fill the anterior chamber and even perforate the sclera. It is one of the symptoms of the tertiary stage of syphilis, is usually met with in only one eye and is found most frequently in the upper and outer part of the iris. When it is formed in the ciliary body it causes a blue black protrusion of the sclera, which may reach the size of a small cherry, attended by severe pain. Occasionally it may undergo involution, leaving a dark, pigmented spot in the sclera, or it may perforate this membrane. Sometimes such a perforation occurs in two places.

When it develops in the iris, either with or without a preceding plastic iritis, it causes an opacity in the neighboring part of the cornea and the formation of many deposits on *Descemet's* membrane. When the tumor breaks down a hypopyon appears in the anterior chamber. The lens may become cataractous and even partially absorbed as the result of the pressure, and secondary glaucoma may be produced, the same as with other tumors.

The prognosis is unfavorable as a rule. Tolerable vision is preserved in at most 30% of the cases, but usually the eyeball atrophies and has to be enucleated. Our patient presents no such clinical picture, and even though it may be impossible in many cases to say whether a papule, or a gumma is present, it is better to make a clinical distinction between iritis gummosa and iritis papulosa.

Papules belong to the inflammations of the deep tissues of the iris. The only exception is the superficial *early papule*, which has no practical importance because it is very small and very rare. In selected cases these minute syphilitic nodules may be seen to develop from the early roseola that has been described already. They usually lie isolated in the anterior layer of the stroma of the pupillary region, are bright red, like the roseola itself, and as a rule disappear quickly either with or without treatment. All other papules of the iris develop beneath the anterior layer of stroma in the deep parts of the tissue.

The papules at the margin of the pupil may be *early* or *late*. The early form is associated with far more œdema and inflammatory hyperæmia than the other, and is therefore redder in appearance. In the late form the clear cell infiltration is more prominent, and therefore the late papules give rather the impression of clear, smooth nodules.

When we examine the anterior surface of the iris of our patient with *Zeiss*' loupe it can be seen to be elevated over both nodules, almost as if it were a sort of vesicle. This appearance has been compared to the pemphigus vesicles found on the skin. In consequence of this elevation of the anterior layer of the iris the vessels that run over the nodes are arched. The thinning of the anterior layer of the iris, as well as the redness and extension of the vessels can be made out clearly. In the early form the œdema may spread and elevate larger vessels, so that the papule may appear larger than it really is. When the accumulation of fluid is sufficiently great it may rupture the anterior layer of stroma and then membranous, firmly adherent deposits of fibrin may for a time cover the real papule. Hemorrhages may also occur in the course of an iritis when the anterior layer of the stroma is thus elevated by œdema.

If the inflammatory œdema about the papule is absorbed either spontaneously, or as the result of therapeutic measures, or if the nodule becomes enlarged as the result of increase of its cell elements, the node presents a gray yellowish appearance; much redness shows that we have to deal with an early papule.

Early papules appear under various forms. In this patient there are two nodes at the margin of the pupil. The number varies, but seldom exceeds six. The reason why they usually appear in the region of the sphincter is that the majority of the capillaries and vessels that are attacked in secondary syphilis lie between the muscle cells and in the pupillary portion of the iris. In some cases the entire pupillary margin is wall like, because the papules elevate it like a red ring. After recovery indentations may be seen between the posterior synechiæ that have been formed. A ledgelike thickening may also develop in the pupillary region, as was pointed out by *v. Michel.*

The late papules likewise appear by preference in the region of the sphincter, but are met with more rarely. They may be single, but when multiple do not coalesce. They may be differentiated from the early papules by their firm consistence and perfectly smooth surfaces. The anterior surface of the iris is not detached over them, the surrounding hyperæmia is less, they are of a gray or yellow gray color, as a rule are larger than the early forms, may extend peripherally far from the sphincter and are not so easily absorbed. Their involution frequently begins with a central softening, which is to be recognized through a wavelike depression in the anterior surface of the iris. The contents may be evacuated into the anterior chamber and form a sort of hypopyon.

Another variety is the cellular, rapidly growing papule described by *Krueckmann* as hemorrhagic infarction. In syphilis of the eye the vessels are chiefly diseased, as can be demonstrated microscopically, hence it can be readily understood that hemorrhages may take place into the anterior chamber when such a papule breaks down. A yellow and a red zone can be distinguished sometimes in such a hemorrhagic hypopyon.

There is no sharp line of demarcation between the early and late papules, for they do not always develop in the same way, and their growth may be rapid or slow, but the attempt to differentiate them is of clinical interest, because generally the firm nodules may be considered indicative that the syphilitic infection is of long standing.

We meet with still other forms of syphilis in the iris. One of the most important is the occurrence of papules in groups. Hitherto the pupillary zone of the iris has been the place of predilection, but papules may develop in other parts, and then we can frequently trace all the stages of development of œdematous and firm nodules in juxtaposition. This *group syphilide* is frequently confined to a single sector of the iris, but the larger nodes come from the ciliary body, while the smaller ones are situated more toward the pupil. The suggestion comes involuntarily that this is a sort of embolism of the agents which has taken place in a certain vessel involving its branches. Such a group syphilide of the iris denotes a grave and neglected syphilitic disease. It is usually met with in elderly and feeble persons and the prognosis is correspondingly grave. Relapses are frequent and later complications on the part of the central nervous system are to be feared.

After they have developed, the papules undergo absorption, especially under treatment. The yellower the color the more rapid is the absorption. A depression is formed at the top of the papule, which gradually becomes flatter. It has been mentioned already that the papules may empty their contents into the anterior chamber and that hemorrhages may occur. A wartlike excrescence of granulation tissue may form in the place of the papule when hemorrhage and excretion of fibrin follow its evacuation; then a red tubercle of granulation tissue proliferates for a time on the iris and absorbs the fibrin.

The most remarkable symptoms in the absorption of the papule are the loss of pigment sustained by the anterior layer of the iris, and the peculiar nature of the posterior synechiæ left in the region of the sphincter. The place where a large papule has been situated in the iris can be recognized from the fact that in that place the anterior layer of the iris has no pigment, but presents a cutaneous appearance through which show the black cells of the retinal pigment layer. This depigmentation is due to the facts that the anterior layer of stroma and the vessels were subjected to a considerable degree of stretching by the papule, and that the chromatophores themselves were injured. When we consider that in syphilis the vessels are seriously diseased and sometimes obliterated, the atrophy of their chromatophore coats is readily understood.

Such spots of discoloration may occur in the iris without a preceding iritis. *Krueckmann* called attention to this and likened the spots to leucoderma. In these cases whitish spots appear during the first weeks or months after the infection in the ciliary portion of the iris, especially in the region of the crypts, with the deep vessels appearing in them as white branches. *Krueckmann* found this symptom to be directly related to the secondary stage.

The second important symptom in the retrogression of the papule of the iris is the atrophy of the muscle cells, which predominates over the depigmentation in the region of the sphincter. At this place the syphilitic disease of the smaller vessels is most marked, and is productive of atrophy of the muscle fibres. We may say that a circle of atrophic places in the region of the sphincter strongly indicates a past syphilis of the iris.

The posterior synechiæ which follow the formation of papules at the pupillary margin have broad bases, are more or less circumscribed, and are composed not only of the retinal pigment layer, but also of the deeper parts of the tissue of the iris.

Statistics show that the papular form appears somewhat sooner after the infection than the diffuse, fibrinous iritis, mainly before the sixth month. Almost three fourths of all the patients are between twenty and forty years of age and those with papular iritis are younger on the average than those with the plastic form. Males are affected more often than females.

Under energetic treatment with inunctions the acute form of fibrinous syphilitic iritis usually recovers in a few weeks. The prognosis for recovery is good on the whole, but with some limitations. Some posterior synechiæ at least very frequently remain. Even the best antisyphilitic treatment may not prevent the outbreak of an iritis. It may appear after discontinuance of the treatment, when all other syphilitic affections of the skin and mucous membranes have disappeared.

Relapses are frequent. According to some statistics they occur in 16% of the cases. Likewise in one half of all cases the vision is more or less impaired. Such relapses cause an increase of the posterior synechiæ and lead to seclusion and occlusion of the pupil with their well known sequelæ; atrophy of the iris, secondary glaucoma, complicated cataract, opacities in the vitreous and phthisis bulbi. The malignancy of the infection and the reduced power of resistance of the organism play their parts. Iritis is looked upon by many authors as a sign of grave syphilis.

The prognosis is generally less favorable in the papular and gummatous forms than it is in fibrinous iritis. The formation of nodules is indicative of a more serious condition. They may heal and leave no trace, or at most discolored patches and posterior synechiæ, but statistics show that almost 50% of the eyes diseased in this manner become totally amaurotic. The chain of disease also is not usually concluded with that of the eye. *Trousseau*, for example, found that of 40 patients who had specific iritis 34 developed later such grave diseases as paralysis and tabes.

Under the microscope syphilitic iritis presents the picture of ordinary chronic inflammation. In the fibrinous form the iris is thickened and infiltrated with round cells, which in great part accompany the vessels. Two symptoms are preeminently characteristic; first the change in the small arterioles, the lumen of which is almost wholly occluded by the proliferation of the intima. Outside the adventitia is a great proliferation of epithelioid cells. Little round nodules, which cannot be perceived clinically, may be seen in the microscopic section of a clinically fibrinous iritis, which proves anatomically that syphilitic iritis is a discrete, deep inflammation of the iris. These circumscribed accumulations of cells are composed of lymphocytes and epithelioid cells, and may contain giant cells. They may be differentiated from tuberculous nodules by the fact that they are richly provided with capillaries and show no signs of disintegration.

49. Wassermann's Syphilis Reaction and Its Importance In Ophthalmology

In our clinical studies we are constantly impressed with the importance of syphilis in many diseases of the eye, and the case of grave plastic iritis that has just been studied shows how necessary it is to determine as quickly as possible whether syphilis is present or not. Then we lose no valuable time trying other remedies and can proceed with antisyphilitic treatment in quite a different manner from that employed when we can calculate only on the possibility of the presence of this disease. The history and physical examination of this patient do not furnish conclusive evidence, but, thanks to the advance which has been made in the study of immunity, we are now able to determine from the serum biologically with practical certainty whether or no he has a syphilitic infection.

I consider it necessary that we should not only appreciate the great importance to ophthalmology of this serum diagnosis, the so called *Wassermann* reaction, but should also be acquainted with the reaction itself. A modern eye clinic which is not equipped to provide this scientific test is not of high standing. It is more convenient, certainly, to send the blood to be examined to a laboratory where such work is done daily, but that does not excuse us from the duty of mastering the method and of being able to demonstrate it. In the short time at my disposal I cannot give a comprehensive review of the serum diagnosis of syphilis as it stands today, with all its mooted scientific questions. Its usefulness and certainty have long been decided, so I shall try simply to make intelligible the experiment by means of which the presence of syphilis is made manifest.

I. The Nature of the Complement Fixation Reaction in Syphilis

When foreign blood corpuscles, like those of a sheep, are injected into a rabbit certain antibodies appear in the blood serum of the latter through an immunity reaction of the body. These antibodies, the hæmolytic amboceptors, withstand a heating to 56°, which renders the serum inactive. If blood corpuscles are introduced into such an inactive serum, which has been rendered specifically immune by blood corpuscles of the sheep, the amboceptors join with the red blood cells, although the latter remain morphologically intact, but as soon as some fresh, not heated, active guinea pig serum, which produces no effect by itself on the blood cells, is added to this mixture the blood corpuscles become dissolved; this is hæmolysis. The bodies contained in the guinea pig serum, which have to be added to the specific amboceptors, are called complement. Hæmolysin is thus composed, according to *Ehrlich*, of amboceptors and complements. To investigate this complement *Bordet* and *Gengou* took an immune serum produced with cholera vibrios instead of blood cells, which contains amboceptors specific only to the cholera bacteria. When these bacteria were brought into contact with the cholera serum these amboceptors seized upon them, and when *Bordet* added our complement to such amboceptors laden with vibrios he found that he could add blood cells and the hæmolytic amboceptors later without exciting hæmolytic action, because the bacteria laden with the specific amboceptors had acquired the ability to appropriate from the hæmolysin (hæmolytic amboceptor + complement) the complement of the guinea pig serum and by this means to frustrate the hæmolysis. *Wassermann's* syphilis reaction is based on this complement fixation. *Gengou* found in immunization that antibodies appear and can be recognized by such a complement fixation not only when bacteria are present, but also when the animals have been treated with dissolved albuminoid substances. In 1905 *Max Neisser* and *Sachs* demonstrated that the origin of albumin taken from any source could be determined accurately with the help of the complement fixation reaction. This may be proved by the following experiment: Take an inactive serum of a rabbit which had previously been treated with albumin obtained from an animal lens; add to it only a trace of lens albumin and the antibodies of the immune serum combine with the lens albumin which caused their formation in the organism and is therefore called antigen. Add again our complement and some time later the sheep's blood amboceptors and the sheep's blood; no dissolution takes place. But the hæmolysis is inhibited only when lens albumin is used and thus the lens albumin can be differentiated from all other varieties. *Wassermann* and *Bruck* then discovered that complement fixation takes place with dissolved extractives from bacteria, the same as with albumin and complete bacteria, and this led the way to the clinical recognition of disease through the blood serum. Now we can either take the bacterial extract and ascertain whether the specific amboceptors that appertain to those bacteria are present in the serum of a patient, thus determining whether he has the specific disease in the course of which those antibodies are formed, or we can make a specific immune serum which contains amboceptors produced by a morbific agent and see whether, when this is mixed with human blood serum, the latter contains any dissolved material of the microorganism in question. Many such experiments have been made. *Wassermann* and *Bruck* found dissolved material from tubercle bacilli in tuberculous organs by the aid of tubercle bacilli immune serum, and conversely demonstrated the specific antibody, the antituberculin, in the blood by the aid of tuberculin. Analogous results have been obtained in other diseases, such as typhoid fever and meningitis. An antigen can be detected in a diseased organ, or its extract, with the help of the complement fixation reaction. This brings us to the reaction of syphilis. *Wassermann*, *Neisser* and *Bruck* made an extract from the liver of a fœtus that had hereditary syphilis, and found that with it the serum of syphilitic monkeys gave complement fixation. They then found that the serum of syphilitic men when mixed with the syphilitic extract deviated the complement, while the serum of nonsyphilitics did not, and thus revealed the great diagnostic importance of the syphilis reaction. Under the influence of the syphilitic infection, i.e., under the influence of spirochætæ, an antibody originates in the blood serum of syphilitics which, together with the extract of the spirochætic material from the syphilitic liver, seizes upon the complement and so prevents hæmolysis by the hæmolysin. If an antibody which possesses these properties is present in the blood of a man, he is syphilitic. But the production of the syphilis reaction is not so simple as this. Spirochætæ were always present in the syphilitic livers from which the extracts were made that gave positive reactions with syphilitic serum, but soon numerous investigators, like *Kraus* and *Volk*, *Weil* and *Braun*, *Levaditi* and others, found that extracts not only of syphilitic livers, but also of normal human and animal organs gave well marked reactions in combination with syphilitic serum, but only with syphilitic serum. *Alcoholic* extracts proved especially efficient and it became evident that the substances in these extracts, the antigen, is present in greater quantity in syphilitic organs. If the antigen necessary for the reaction can be obtained from both syphilitic and normal organs it cannot be that *Wassermann's* reaction depends on the combination of a specific syphilitic antigen and a specific syphilitic amboceptor. Numerous investigators, such as *Landsteiner, Meier, Porges, Wassermann, Levaditi,* and *Mueller* among others, found this to be the case, and that particularly alcoholic extracts from syphilitic and normal organs gave the reaction in connection with syphilitic serum, and it was concluded that *lipoid* rather than albuminoid substances take part in *Wassermann's* reaction. Many attempts have been made to take from complement fixation the character of an immunity reaction and to give it a purely physico chemical explanation. A number of lipoids, such as lecithin, sodium oleate, the biliary salts and cholesterin, react positively with syphilitic serum. According to *Porges* and *Meier* syphilitic serum is also able to cause precipitates in emulsions of lecithin and other lipoids. Such precipitates have also been observed after the addition of distilled water to syphilitic serum. The theory is advanced, based on the researches of *Seligmann, Sachs,* and *Altmann*, that the syphilis reaction is a colloid precipitation between hydrophile colloids and albuminoids which are closely related to globulins, which creates a greater zone of precipitation in the syphilitic serum in consequence of a diminished stability. Other investigators, like *Wassermann* and *Bruck*, still maintain that it is an immunity reaction. This theory is favored by the result of *Bruck's* experiment in which he was able to obtain material in the serum of monkeys that had been previously treated with syphilitic extract, which gave complement fixation. *Weil* and *Braun* add that in syphilis a disintegration of tissue takes place in which autoantibodies are formed, while substances are set free by the disintegration, and that the material recognizable through this syphilis reaction portrays these products of reaction in comparison with the proper cell constituents of the body. *Weil* and *Braun* also claim that the principal part is played by the albumin complex in this combination of albumin and lipoid instead of the antibodies. At any rate, under the influence of the agent of syphilis a material normally present in the body undergoes a marked increase, and the extracts from syphilitic organs are always more serviceable than those from normal organs. Hence there are those who believe that the syphilis reaction contains not only the nonspecific factor of the action of the autoantibodies upon albumin and lipoid, but also a specific factor, syphilitic amboceptor + syphilitic antigen. Preeminent among these is *Wassermann* himself.

II. The Technique of Wassermann's Reaction

However these purely theoretical questions may be decided in the future the practical value of *Wassermann's* reaction has been established by many thousands of tests. We need for its performance: 1. Serum from a patient in whom syphilis is suspected and serum from a healthy man. The serum is obtained best from the ulnar vein. A portion of both serums is inactivated by heating it to 55°. 2. An extract from the liver of a syphilitic fœtus as antigen. One gram of this liver is cut into fine pieces, placed in 9 grams of absolute alcohol for twenty four hours, the solution being constantly shaken and afterward filtered. This extract may be kept for a long time, but its efficiency must be tested first with a number of serums. It may be considered useful when a comparatively small amount produces complete inhibition of hæmolysis in an unquestionably syphilitic serum, while it does not affect the complete solution of the blood in a normal serum, when in a double dose alone it does not inhibit hæmolysis, and when alone it does not produce hæmolysis. The effective quantity of our extract, which fulfills these requirements, is 0.2 ccm. In order to be independent of these difficulties in the preparation of the extract and to be able to secure equally good results, *Sachs* and *Rondont* have devised a very useful artificial mixture of sodium oleate, lecithin, oleic acid, water and alcohol. 3. A hæmolytic amboceptor. This is obtained from the serum of rabbits into the veins of which sheep's blood corpuscles have been injected. 4. A 5% suspension of sheep's blood corpuscles in an 85% salt solution. This blood must first be washed several times. 5. Serum obtained from the blood of a normal guinea pig. This guinea pig serum contains the complement upon the deviation by which of the hæmolytic amboceptor the success of the experiment depends. At the beginning of the experiment 0.05 ccm of guinea pig serum (complement) is placed in a number of test tubes, to which is added in diminishing quantities rabbit immune serum (amboceptor) and then blood, in order to determine the exact quantity of amboceptor necessary for complete hæmolysis. Three or four such amboceptor units are used in the principal experiment. In this principal experiment test tube No. 1 contains 0.2 of inactive serum of our patient + syphilitic extract + 0.05 of guinea pig serum; this mixture is allowed to stand in the incubator for an hour and then a fourfold quantity of amboceptor and blood is added. Hæmolysis is completely inhibited. On the other hand, neither the normal serum with the syphilitic extract, nor the syphilitic serum alone have fixed the complement. From this result we may conclude that our patient has syphilis. Some other control experiments are necessary. We must have at our disposal a positively syphilitic serum, and we must examine the serum of the patient both in an active and in an inactive condition. I present here a table delineating the principal experiment.

The Wassermann Reaction

Tube No.	Inactive Syphilitic Serum.	Guinea Pig Complement.	Syphilitic Extract.	Normal Human Serum.	Addition after an Hour of		Result.
					Hæmolysis Amboceptor.	5% Suspension of Blood.	
I. . .	0.2	0.05	Single dose	—	Fourfold dose.	1.0 ccm of the suspension.	Inhibition of the hæmolysis.
II. . .	0.2	0.05	—	—	"	"	Solution.
III. . .	0.4 double dose	0.05	—	—	"	"	"
IV. . .	—	0.05	Single dose	—	"	"	"
V. . .	—	0.05	Double dose.	—	"	"	"
VI. . .	—	0.05	Single dose	0.2	"	"	"
VII. . .	—	0.05	—	—	"	"	"

III. The Importance of Wassermann's Reaction in Ophthalmology

The question arises: Of what value is the reaction to us in ophthalmology? This is not difficult to answer. We need only to institute the reaction in a great number of eye diseases and compare the clinical diagnosis with its outcome, as has been done by *A. Leber.* He found the reaction to be positive in a very high percentage of the syphilitic diseases of the eye, even when years had elapsed since the infection. The reaction is the same whether the disease of the eye is due to hereditary, or to acquired syphilis. Our knowledge concerning the value of *Wassermann's* reaction is still incomplete, but, according to *A. Leber*, between 80 and 90% of the patients with secondary or tertiary syphilis who have positively syphilitic eye diseases, and 90% of those with diseases of the eye due to hereditary syphilis, react positively. We can conclude from a positive reaction that either a manifest, or a latent syphilis is present in the organism, and this is a very great diagnostic advance in ophthalmology. It is self evident that a positive reaction does not always indicate with certainty that the disease in the eye is syphilitic, for a syphilitic may suffer from a tuberculous iritis or scleritis, for example, but such occurrences are not very frequent, although we are not yet accurately informed in regard to them. Sole dependence should not be placed on the outcome of the reaction, but each patient should be submitted to a thorough clinical examination. A negative reaction does not always prove the nonexistence of syphilis. As a rule a positive reaction can be obtained first in the sixth week after the infection, but this incubation period rarely needs to be considered in ophthalmology, because most of the syphilitic diseases of the eye belong to the secondary stage. The positive reaction must be looked upon as evidence of the generalization of the virus, but it has been shown repeatedly that it is less apt to be obtained in the latent than in the manifest stage, and is demonstrable on the average in 50 to 60% of the cases. Finally the result may be negative because the substances necessary for the reaction have disappeared from the blood under the influence of specific treatment. These remedies exert no influence on the substances themselves, their effect depends much more probably on the enfeeblement or destruction of the virus, which allows a new production of these substances to take place throughout the body. Hence a disappearance of the reaction has been accepted as a therapeutic guide by *Neisser*, *Bruck* and others. It is useless to estimate titrimetrically and record the degree of reaction, as has been done by *A. Leber*, for, as he himself demonstrated, no conclusion can be drawn in regard to the severity of the disease from the degree of reaction. We must be content to note the positive or negative result, without consideration of the degree. Only the disappearance of the reaction in syphilitic diseases of the eye under the influence of treatment is of interest to us.

50. Uveitis Tuberculosa and the Importance of the Tuberculin Reaction and of Tuberculin Therapy in Ophthalmology

In clinical instruction we have holidays and work days; sometimes we have to deal with the daily monotony, sometimes with cases that captivate us. The day may be said to belong to the former class on which we study the pernicious disease of the human race, tuberculosis, in the eye.

I. The Clinical Picture of Iritis Tuberculosa

I do not present these three patients together without a reason. In the peripheral portion of the iris of one of them are scattered a number of gray yellow nodules, which vary in size from that of the point to that of the head of a pin, and evidently contains few, if any vessels. The details of the iris are obscured and some posterior synechiæ are met with.

In order to understand this clinical picture of tuberculous iritis it must be remembered that the tuberculous nodules develop principally in the walls of the arterioles, and that therefore the region of the sphincter, which is supplied with capillaries, often remains comparatively free from their presence, in contradistinction from the syphilitic diseases, in which the region of the sphincter is the place of predilection for the formation of papules. In many cases the minute nodules occupy the pupillary region also, and the sight of these disseminated nodules is always suggestive of tuberculosis.

Tuberculosis of the iris may present other clinical pictures. In our second patient we find a slow, chronic iritis. No individual nodules can be seen, but the entire iris appears thickened, while its anterior surface seems swollen, elevated in some places, depressed in others. The pupil is secluded and the general appearance is almost like that of a pound cake. In such a case the entire iris seems to be changed into a sort of granulation tissue.

Another form of tuberculosis of the iris is characterized by a solitary tubercle at the angle of the anterior chamber, which may grow until it has filled the chamber (see illustration), and may even perforate the eye. In such a case the sclera in the region of the ciliary body gradually becomes thin and more and more ectatic, until a scleral or corneal staphyloma is formed; perforation finally takes place, followed by phthisis bulbi. In all such cases the diagnosis of tuberculosis of the iris is the first to be considered. To *v. Michel* is due the credit of having been the first to appreciate the important part tuberculosis plays in diseases of the eye, and of having called attention urgently to the fact that it may be present in such diseases without producing a typical clinical picture. Such a condition is exemplified by our third patient, who has an obstinate scleritis associated with an iritis that runs a slow, lingering course. *Stock* has furnished excellent support to the theory promulgated by *v. Hippel*, that tuberculosis is a common, atypical dis-

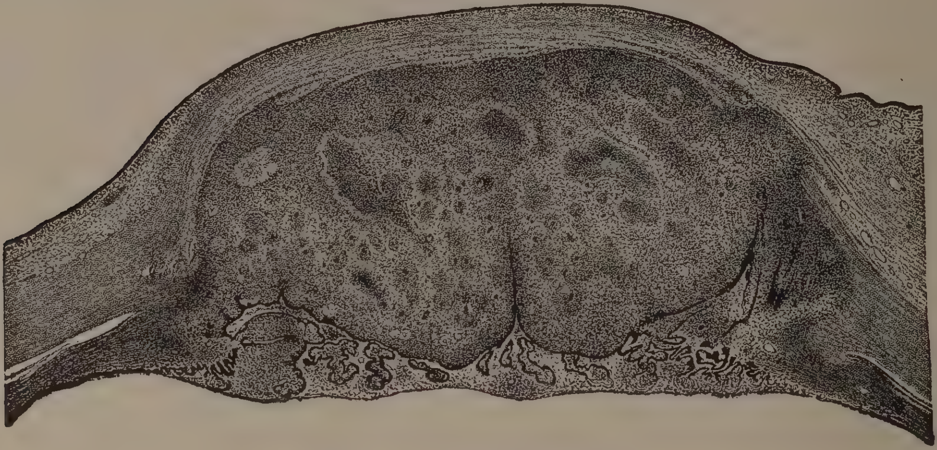


FIG. 31.—Iritis Tuberculosa.

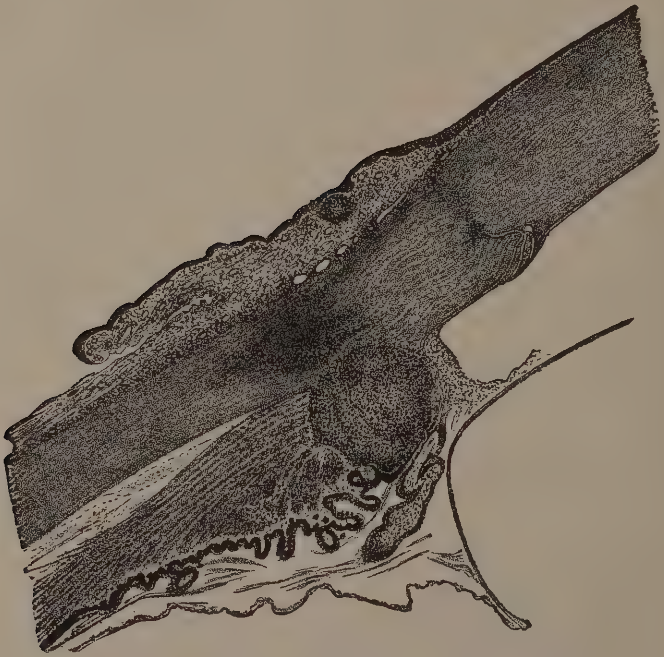


FIG. 32.—Tuberculosis of the Ciliary Body.

ease of the eye. By intravenous inoculations of tubercle bacilli in rabbits he produced changes in the eye which were very similar to those of the chronic uveitis observed in man. A chronic iritis with nodules and a cyclitis may be produced by the same means, as well as a disseminated chorioiditis, whence it may be concluded that many cases of disseminated chorioiditis in man are of tuberculous origin. *Stock* also produced a *scleritis*, a *sclerotizing keratitis, little nodules in the conjunctiva* and a chalazion like tuberculosis of the tarsus. Finally he proved by the pathological examination of eyes which had been destroyed by chronic iridocyclitis that tuberculosis is the cause in many of these cases. The part played by tuberculosis is much greater than is to be expected at first from the clinical picture.

II. The Diagnostic Value of the Tuberculin Test

Such cases and such experimental facts are convincing that tuberculosis must be thought of in connection with diseases of the eye which do not appear to be tuberculous at first glance, and the question is how to make a positive diagnosis in the absence of a good clinical picture.

A tuberculous infection may be demonstrated anatomically by the characteristic structure of its tissue, or by inoculation of an animal with the tuberculous substance, or by staining the tubercle bacilli. This can be done occasionally in diseases of the eye, but only in a limited number of cases. A diseased lacrymal sac, a nodule from the conjunctiva, or a portion of the episclera may be investigated in these three ways, but diseases of the cornea, of the iris and especially of the fundus, cannot. It has also been proved repeatedly that the histological condition is not always typical, that often bacilli are hard to find, and that an inoculation may prove negative when the diseased substance is only slightly virulent. These defects enhance the importance of the tuberculin reaction in ophthalmology. The great achievement of *Robert Koch* has supplied us with a means by which we can learn through a specific reaction if active or latent tuberculosis is present in the eye. This means is old tuberculin. The directions given by *Koch* himself in regard to the diagnostic use of old tuberculin are as follows:

"First the temperature of the patient is taken for one, or better two days, in order to determine that it does not rise above 37°. Patients with temperatures of over 37° are unfit for the diagnostic use of tuberculin and the test should under no circumstances be made upon them. When the patient has been found to be a fit subject an injection of from 0.1 to 1.0 mg of tuberculin is made subcutaneously during the forenoon; in feeble persons we begin with 0.1 mg, but in robust individuals with presumably slight tuberculous changes, the initial dose may be 1.0 mg. If no rise of temperature follows the first injection, a double dose should be given not on the next, but on the third day. If there is a rise of temperature, even though it be so slight as ¼ of a degree, the dose is not increased, but the same quantity is injected again, after the temperature has returned completely to normal. It happens very often that the reaction which follows the second injection is much more marked than that after the first, although the dose is the same. This is a particularly characteristic symptom of the action of tuberculin and may be said to be an infallible sign of the presence of tuberculosis. When no reaction follows the first small doses the quantity may be increased to 5 mg, and finally to 10 mg. For the sake of certainty I am accustomed to give the latter dose twice and then, when no reaction follows, I consider myself justified in the assumption that no fresh, or advancing tuberculosis is present which needs a specific treatment."

More recently two new methods have been added, the cutaneous inoculation of *v. Pirquet*, and the conjunctival instillation of *Wolff-Eisner* and *Calmette.*

In *v. Pirquet's* cutaneous reaction the skin of the forearm is scarified, as in vaccination, in three places near each other. Two of these scarifications are left as controls, while a drop of a 25% solution of tuberculin is placed upon the third. If an infection of tuberculosis is present in the organism, a characteristic wheal develops at the place of inoculation. This reaction is so sensitive that it is positive not only when active tuberculous foci are present, but also when the lesions are old and extinct. As such extinct tuberculous lesions are exceedingly common in adults, the cutaneous inoculation of *v. Pirquet* is useful chiefly in childhood. When the test is negative in an adult, it is fairly certain that that person is free from tuberculosis. As the test is easily performed and is devoid of danger, it is used a great deal to determine quickly whether tuberculosis is present in the organism.

In the conjunctival reaction a drop of a 1% solution of old tuberculin is instilled into the conjunctival sac, where it excites a local reaction, if active tuberculosis is present in the organism. An extremely interesting phenomenon is associated with this test. According to *Wolff-Eisner* a positive conjunctival reaction indicates active tuberculosis and its occurrence arouses suspicion of the disease in a clinically healthy person. If the test proves negative in the presence of manifest tuberculosis, the negative result indicates a bad prognosis. Unfortunately the usefulness of this tuberculin test in ophthalmology has proved very limited, for, even when it is employed carefully and properly, it may excite severe reactions in healthy eyes, and cause grave injury. Above all too great a reaction is to be feared in an eye in which the presence of tuberculosis is suspected from the existence of a keratitis interstitialis, chorioiditis disseminata, iritis chronica, or a grave form of keratoconjunctivitis eczematosa. *Wolff-Eisner* himself excludes as unfit for this test all eyes which are known, or suspected of being tuberculous. Therefore it is not permissible that this test should be used for the diagnosis of ocular tuberculosis. I cannot advise the use of the conjunctival reaction at all, because frequently it is not easy for the practicing physician to determine whether a contraindication on the part of the eye is present or not. Ophthalmologists themselves are still in doubt clinically concerning the tuberculous etiology of many ocular diseases and have to base their diagnoses on the results of the tuberculin tests, and naturally we choose the tests from which no injury to the eye itself is to be expected. Finally an antituberculin can be demonstrated in the serum of persons with tuberculosis after the manner of *Wassermann's* reaction. But in contrast with the great importance of the syphilis reaction this complement fixation method is of no use in ophthalmology. Aside from the complicated technique the results are inaccurate and scarcely comparable. Besides, the presence of antituberculin in the serum tells nothing concerning the cure of the disease, for tuberculin immunity is not the same as the immunity of tuberculosis. For diagnostic purposes *v. Pirquet's* and *Koch's* reactions alone are to be considered in ophthalmology. First we try *v. Pirquet's* cutaneous inoculation. As this is a very sensitive test and gives a positive result in most adults, a negative result enables us to exclude tuberculosis with a fair degree of certainty, but, for the sake of safety, we frequently employ *Koch's* injection of old tuberculin, which still remains the chief diagnostic method. Only we must understand clearly what diagnostic conclusions can be drawn from it. If the subcutaneous reaction is negative, the indication is that the ocular disease is not tuberculous. If a local reaction takes place in the eye, either alone, or in connection with a general reaction, the ocular disease may be considered tuberculous, even when not suggested clinically. But if the test is followed by a general reaction alone we cannot ascribe a tuberculous nature to the disease in the eye without further evidence. It is quite possible for a patient who has a tuberculous lesion somewhere in the body to have an iritis due to some other cause. When the general reaction is positive, and there is no local reaction, it may be assumed that the ocular disease is probably tuberculous only when positive signs of other disease are not demonstrable, and when a careful physical examination has left no ground for the supposition that the reaction is to be referred to another tuberculous lesion in the body.

III. The Therapeutic Value of Tuberculin Treatment in Ocular Tuberculosis

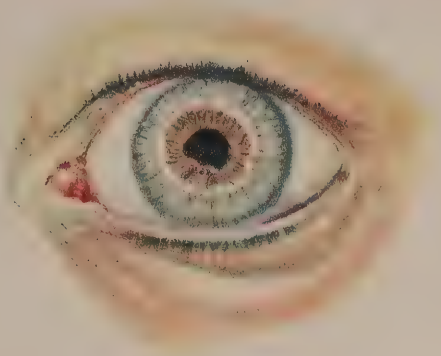
Ophthalmology has had to share the bitter disillusion that followed the mishaps of the tuberculin therapy of pulmonary tuberculosis. If there is a disease for which a specific therapy is in constant demand, it is tuberculosis. This is true also of tuberculous diseases of the eye, and it is to the credit of *v. Hippel* that he has proved his faith in the possibility of utilizing the specific remedy in this organ. After years of observation he concluded that tuberculin was a remedy for ocular tuberculosis, and his statements have been confirmed repeatedly. Yet the complicated conditions incident upon recovery from tuberculosis demand more attention, and too much must not be expected from the tuberculin treatment. My own judgment, concerning the therapeutic value of tuberculin in the treatment of ocular tuberculosis, is that the employment of a specific remedy in these tuberculous diseases is a logical consequence of the study of immunity, and that the use of tuberculin in those diseases of the eye which we have hitherto been obliged to leave to the doubtful efficiency of other remedies, or more correctly to spontaneous recovery, is beyond doubt an advance. I am convinced that this sort of active immunization promotes recovery from ocular tuberculosis in many cases. *But I am also convinced that the therapeutic capacity of tuberculin in the treatment of ocular tuberculosis has been, and still is much overestimated.* Such uncertainty in the estimation of the value of this remedy may be accounted for in several ways.

In the first place opinions are still divided in regard to the way of using it and as to the dosage. While some ophthalmologists use only minute doses in order to avoid fever and focal reactions, *A. Leber* declares that focal reactions are unavoidable as expressions of its specific influence, and are therefore indispensable for its therapeutic effect.

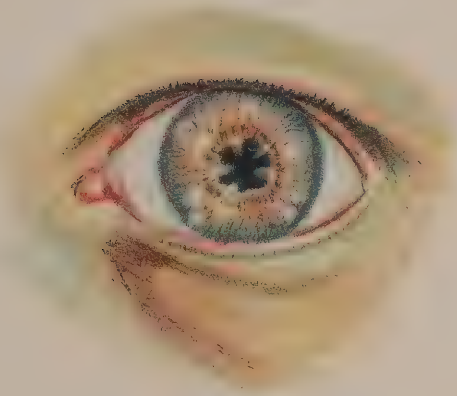
In the second place it has not yet been ascertained which tuberculin is best suited for the purpose. *v. Hippel* explicitly claims that permanent recovery in many of his cases, which had relapsed, could be attained only through the new tuberculin, T. R., but these cases had in fact been inoculated with old tuberculin. While many have faith in T. R., the sentiment is growing that we should return to old tuberculin, a preparation which is a priori far less objectionable. *A. Leber* says truly that, in view of the conditions favorable to involution in ocular tuberculosis, the use of old tuberculin is well founded, as it may prevent toxic injuries to the eye by the formation of an immunity to the poison.

In the third place the factor of spontaneous recovery is one which it is extremely difficult to estimate, when dealing with ocular tuberculosis. The experiments of *Stock* have proved incontestably that, in comparison with other manifestations of tuberculosis, the lesions in the eye are comparatively benign, a fact which is constantly brought to the attention of everyone who studies these diseases critically. This comparative benignity is so evident that many ophthalmologists, like *Leber* and *Wagenmann*, speak properly of a weakened tuberculosis. If tuberculosis in the eye is so benign in many cases that elaborate biological reactions are necessary to establish it as only the probable cause of the lesions, it is self evident that it must be extremely difficult to determine how far recovery can be ascribed to the treatment with tuberculin. Improvement can be attributed to it with some degree of probability only in cases in which other therapeutic measures have failed. Such cases have convinced me that an advance, though only a slight one, has been made in the treatment of ocular tuberculosis by its introduction. Still another point has to be taken into account in passing judgment on this form of treatment. When a patient with a benign form of ocular tuberculosis enters the clinic, and remains for months under entirely different conditions as regards bodily rest, care and food, in addition to the treatment with tuberculin, we can hardly credit the therapeutic result wholly to the tuberculin.

I am one of those who believe in the study of immunity, and I have always taken the stand that advance in therapeutics must be made along the lines of specific, etiologic therapy. Tuberculin treatment of ocular tuberculosis is such a specific therapy, but we must not close our eyes to the fact that mishaps do not cease to occur under this form of treatment. During the past year I have had to enucleate five eyes on account of ocular tuberculosis. Among them were three which had previously undergone thorough tuberculin treatment and had been considered brilliant results confirmatory of *v. Hippel's* statements. *A. v. Hippel* himself has had cases in which this therapy failed. This treatment has its limitations, and the investigators of tuberculosis are continually seeking for new, efficient remedies. Tuberculin treatment may fail not only in grave cases, but even in those of moderate severity; it is unable to prevent the onset of tuberculous disease in the other eye after the lapse of weeks. Its prophylactic, immunizing effect is slight as regards diseases of the eye. It is also unable to prevent relapse in the eye first diseased. The much lauded curative effect of this form of treatment is very limited. *A. Leber* says: "With the difficulty of determining in certain cases whether a recovery is spontaneous, or due to tuberculin, it must be said that the results from the use of old and new tuberculin do not appear to be as good as those obtained by *v. Hippel.*" Still we must be thankful for even the slight advance gained in the treatment of ocular tuberculosis by the remedy presented to science by *Robert Koch.*



a Iritis luetica.



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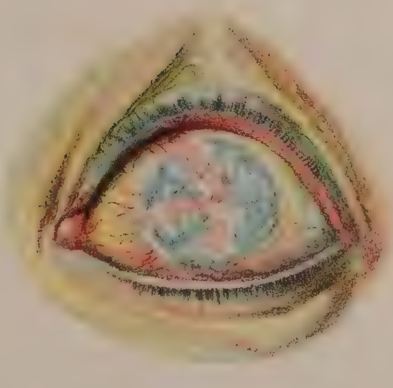

c Iritis and keratitis tuberculosa.

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IV

Diseases of the Lens

51. The Nutrition of the Lens

We have already learned to recognize a number of clinically perceptible physiological changes which take place in our lenses during life by the comparison of lenses taken from young and elderly persons. When we come to study the pathology of the lens we need information concerning its nutrition, for most of its diseases depend on nutritive disturbances of its cell elements. Our knowledge in regard to this subject is still very meagre, and great difficulties lie in the way of its investigation. Science has not yet solved the problem of the nutrition and metabolism of the individual cell. The lens lies in the eye, a masterpiece of living, transparent protoplasm, composed of skillfully united epithelial cells and fibres, enveloped in a delicate capsule, destitute of bloodvessels, and laved only by the intraocular fluids. It is evident that the lens receives nourishment and has metabolism, for it has two obvious functions to fulfill.

It has to remain clear and transparent in order to direct the course of the rays of light correctly and uninjured to the retina. It also continues to grow until very old age. Throughout life new fibres and substances are continually formed from the epithelium of the capsule. The preservation of its normal condition in youth would not suffice for this, a productive nutrition is necessary.

Two procedures are at our disposal for the study of the nutritive conditions of the lens. We may either place the lens in a test tube along with the substances to be investigated, or we may observe its behavior *in situ* and *in vivo*. Many unjustifiable conclusions concerning the nutrition and metabolism of the lens have been deduced from experiments in the test tube, so its attitude toward foreign substances is to be preferred; but it must be understood that we can learn very little about the *nutrition* of the organ from such experiments, because the lens does not live on coloring matters. If the substances used are indifferent to the lens they are either not taken up by it, or are extruded again without having been assimilated, provided the conditions necessary for diffusion are present. Substances which are not indifferent are apt to act as poisons to the lens, and nothing can be learned through them concerning its normal nutrition.

The essential disturbance of the natural conditions of the organ, and the fact that the lens soon dies when outside of the living eye, limit the value of all experiments on the lens in the test tube. In this respect the lens is extremely sensitive to the way in which it is prepared, the temperature and the composition of the surrounding fluid. The commencement of the death of the cell elements of the lens in the test tube is indicated 1, by the dissolution of the connection between the epithelium and the capsule; 2, by the inability of the lens to resist the entrance of water, which increases its weight and volume; 3, by the constant passage outward of albumin through the capsule. The death of the lens begins in different temperatures at different times, according to my observations. On the average it may be said that death of the outer cortical layers is perceptible in from seven to ten hours when placed in a suitable salt solution at the temperature of the incubator. In the ordinary temperature of a room a lens will live somewhat longer, but even in the temperature of a refrigerator it will hardly live longer than twenty four hours. All observations agree that the metabolism of the lens is extremely slow and it is evident that little can be learned concerning the nutrition of the lens from experiments in test tubes, since it dies so quickly under such conditions.

Unfortunately the limitations of the experiments *in vivo* are also unpleasantly narrow. The same objections apply to experiments with foreign substances as to those in the test tube. In addition to this, the organism tries so promptly to rid itself of all foreign substances that nothing can be seen of their effect upon the lens, and their conduct within the lens is wholly unknown. *Yet the nutrition of the lens depends upon its relations to the substances contained in the normal blood and the normal fluid in the eye.* These relations must be understood better before the nutrition of the lens can be investigated.

Four things must be known before this mystery can be penetrated. We must know the exact composition of the intraocular fluid upon which the lens has to live, we must know the power by which the lens appropriates its nutrient substances, we must know what substances it takes from the intraocular fluid, in what proportionate quantities and in what time it appropriates them, and finally we must know accurately the constructive and destructive activity of the living protoplasm of the cells of the lens. None of these problems have been solved satisfactorily as yet, our knowledge in regard to them is simply a patchwork, but I will try to demonstrate the little we know concerning the nutrition of the lens.

I. Composition of the Intraocular Fluid

A. Inorganic Substances

The human eye contains between 0.2 and 0.3 ccm of aqueous. This is a clear, watery fluid which has a specific gravity of from 1.006 to 1.009, has a distinctly alkaline reaction, and contains about 1% of such inorganic substances as sodium chloride, sodium carbonate, potassium chloride and alkaline sulphates, phosphates and earths. The organic substances thus far found in it are, in addition to albumin, which will be spoken of later, small quantities of glucose, urea, paralactic acid and a saccharin producing enzyme. The macroscopic appearance of the aqueous is quite analogous to that of the fluid of the vitreous.

The latter is a perfectly clear fluid which has a distinctly alkaline reaction. Its chemical composition is nearly the same as that of the aqueous. It contains about 1% of inorganic substances together with small quantities of organic substances in the form of sugar, urea and paralactic acid, as well as a substance which resembles mucin and is known as hyalomucoid. Its index of refraction is the same as that of the aqueous, about 1.337. Thus the aqueous and the fluid of the vitreous have a very similar composition, and both contain some oxygen and carbonic acid.

When we compare the composition of the intraocular fluid with that of the blood serum we find that, while the statements concerning the amount of salts contained in the blood vary in the different textbooks on physiology, the amount is approximately the same as in the aqueous.

B. The Albumin Contained in the Intraocular Fluid

Although there is no great difference between the amounts of salts contained in the serum and in the intraocular fluid, there is a striking difference in the amount of albumin each contains. Serum contains about 8% of albumin, the normal aqueous only about 2%. The difference exhibited between two rows of test tubes in which dilutions of serum and of aqueous are treated with picric acid is perceptible at once.

If we assume that serum contains 8%, and aqueous 2% of albumin, then 0.2 ccm of blood serum contains 0.016 gram of albumin, and 0.2 ccm of aqueous 0.00004 gram, i.e., the aqueous contains 400 times less albumin than the same quantity of blood serum. Even though the proportion varies somewhat in individual animals and persons the difference remains very great.

The normal aqueous does not coagulate. It contains no fibrinogenous substances, and probably the other generators of fibrin are either absent, or to be found in only inefficient traces. Serum globulin and serum albumin seem to be of about equal amount in the small quantity of albumin mixture met with in the aqueous. The fluid of the vitreous likewise contains only the same small quantity. It may be demonstrated that there are antibodies connected somehow with the serum albumin, derived albumins equipped with strong chemical affinities, which are retained in the blood by the secretory organ of the eye and are not admitted into the intraocular fluid.

Normal ox serum, even in small quantities, dissolves the red blood corpuscles of the guinea pig by the aid of a specific cytotoxin, the hæmolysin, but in the aqueous and the fluid of the vitreous the blood cells remain perfectly intact. Even when greatly diluted the serum agglutinates blood cells and bacteria. Nothing of this normal agglutination is found in the intraocular fluids. The serum also contains an antitoxin to a toxin, tetanolysin, which protects the blood cells from the poison. This antitoxin is not present in the aqueous and the fluid of the vitreous.

Although in immunization the antibodies are considerably increased in the serum the secretory organ of the eye excludes all bodies with cytotoxic actions from the intraocular fluids, so that only traces of antibodies can be met with in them. I have named this phenomenon the law of cytotoxin retention by the secretory apparatus of the eye, and I think that it must have a meaning.

An increased invasion of such derived albumins might be injurious optically to the eye, but this is hardly probable, because when the albumin in the aqueous is increased, as after subconjunctival injections of salt solution, the vision does not appear to be affected. Or these bodies may be superfluous for the nutrition of the lens. This is just the case, for they are not to be found in the normal intraocular fluid, or in the eyes. Under certain circumstances such bodies with cytotoxic qualities might be injurious perhaps to the lens. In that case the secretory organ of the eye has an important duty to perform for the preservation of the lens. However this may be, there is still more to be learned concerning the derived albumins that help to form the normal nutritive fluid of the lens. We do not know whether the secretory organ of the eye allows them to pass from the blood into the aqueous and vitreous, or whether they are remodelled and prepared for the lens, as foreign nutritive albumins are reformed into albumin of the body in the intestines; we can only test whether they give the same biological reactions as the serum albumin. We find then that the albumin of the aqueous and vitreous reacts exactly the same as the albumin of the serum of the same animal.

Another important question in the nutrition of the lens is that of the osmotic pressure of the intraocular fluid and its relations to the osmotic pressure of the blood. Here we meet with contradictions. Some ophthalmologists believe that the aqueous and the fluid of the vitreous have a higher osmotic pressure than that of the blood serum, and that the lens has continually to contend against a difference of osmotic pressure.

Repeated attempts have been made, especially by *Peters*, to ascribe an important influence upon the origin of different forms of cataract to insignificant fluctuations in the salts contained in the aqueous. On the other hand, *Hamburger* believes that while the aqueous may be a hyperisotonic fluid as compared with the blood serum, the lens is in osmotic equilibrium with this, its natural medium. This question has now been settled. *Nuel* found that the osmotic pressure of the aqueous and vitreous is not higher, but the same as that of the blood. I then demonstrated that the physiological fluctuations of the osmotic pressure of the blood serum, which are by no means inconsiderable, are continually transmitted to the intraocular fluid, and that the lens is adapted to these fluctuations just as well as the blood cells.

If the osmotic pressure of the blood serum is increased by loss of water from the body this fluctuation is transmitted to that of the intraocular fluid, though the equilibrium is established gradually, so that, at any given moment, the osmotic pressure of the intraocular fluid may be either higher or lower than that of the blood serum, but usually both are alike. My pupil, *Rissling*, has studied this subject extensively.

According to the results of my investigations a salt solution which corresponds in concentration to the osmotic pressure of the blood serum is the best isotonic solution for the lens. For ox lenses such a solution contains from 0.9 to 0.95% of salt. While they remain alive these lenses appear faultless and do not increase in weight.

As to the theory that an insignificant increase of the salt in the aqueous may cause a disturbance of the metabolism in the lens and the commencement of a cataract, the latter is certainly not the case. If it were so our lenses would be in danger after loss of water from the body, for example, by forced marches. But such a fear is groundless. A transient, physiological increase of the concentration of the salt is of no importance to the lens, and a permanent increase in the aqueous is impossible because of the law of osmosis, which naturally effects the necessary exchange with the blood. Nothing justifies a theory which asserts an exceptional position for the lens in regard to its relations with the fluid which surrounds it.

If we are correct when we consider the blood serum to be the best isotonic solution for the blood cells, although its osmotic pressure constantly undergoes considerable fluctuation, we must acknowledge that the lens is in osmotic equilibrium with the intraocular fluid, the osmotic pressure of which is no greater than that of the blood. As the lens remains sound in spite of the well marked fluctuation of the osmotic pressure in the blood and intraocular fluid it is evidently adapted to these fluctuations, the same as the blood cells. We shall return in another place to this comparison with the blood cells; here it is of interest because both they and the lens obtain their nutriment through osmotic exchange with the surrounding fluid.

Just as the protoplasm of the blood cells forms a membrane which interferes with diffusion, in that it prevents the entrance of water and the escape of hæmoglobin, so the lens is provided in an analogous manner with a protective device which limits diffusion and guards against the entrance of water. It is a question whether this is an attribute of the lens capsule, or of the epithelium. I am convinced that it is an attribute of both.

It has been proved that in various forms of cataract large areas of the epithelium of the capsule are destroyed, and that this results in an increased influx of water and an enlargement of the lens. This may be easily proved by experiment. The lens of an animal may be massaged through the cornea so as to destroy the epithelium of its capsule while still in the eye. If it is then removed and placed in an isotonic salt solution, together with an uninjured control lens, it can be seen in a few hours that the anterior capsule has become elevated and that water has entered, although the control lens remains perfectly clear and intact.

Against the protective action of the epithelium it has been urged that the posterior capsule has no epithelium and yet the lens is protected against the fluid of the vitreous, but it must be remembered that the lens fibres are inserted in the posterior capsule with broad ends, forming a mosaic, which acts like epithelium. In addition to this, no fluid can enter the lens easily from the vitreous, so that an isolated injury of the posterior capsule is followed usually by only a little cloudiness of the lens.

It cannot be disputed that the lens capsule itself also takes part in the protection of the lens by impeding diffusion. If a small wound, that remains open, is made in the anterior capsule, many more lens fibres become swollen, opaque and disintegrated through the entrance of the aqueous, although little epithelium has been injured. It would be impossible for a total cataract to develop after an insignificant injury to the capsule, if the lens fibres and cells themselves were provided with a sufficient protection against diffusion, so it is evident that the product of the epithelium, the capsule, performs to a certain degree the duty of protecting the lens fibres, perhaps because nutrition can be better assured thus than would be possible if each fibre were supplied with a cell membrane to impede diffusion.

II. How is Nutritive Material Appropriated by the Lens?

First, filtration currents may be excluded. It has been disputed for a long time that fluid may pass through the lens capsule by filtration when there is a difference in pressure; the latest researches of *Leber* render it probable, but this does not hold good for the lens *in the eye*. When the lens is injured the capsule gapes. The hydrostatic pressure in the lens can be only slightly greater than that about it, so when it swells, or a cataract develops, some fluid might be expressed through the capsule, but none could enter.

The lens depends rather on diffusion with the surrounding intraocular fluid for its nourishment. No pores exist in the capsule which can be found anatomically, so the question arises whether it is permeable as such and, if so, for what substances. Experiments have proved that quite a number of substances can diffuse through the isolated lens capsule, including such crystalloids as sodium chloride, ferric chloride, fluorescin, potassium ferrocyanide, eosin, carmin, and methyl blue, and such colloids as egg albumen, serum albumin, hæmoglobin and the colloidal solution of gold, while there are other substances, e.g., Prussian blue, to which the capsule is impermeable. The rapidity with which the individual substances become diffused varies greatly.

The results of these experiments are quite different when the entire lens is used. Carefully prepared, fresh lenses may be placed for several hours in various staining solutions; then, if they are washed off and cut in two, it can be seen that none of the stains have entered them. Such substances cannot penetrate to the fibres until the lenses die.

All such experiments have shown in addition that a diffusion into the lens of such substances as fluorescin, potassium ferrocyanide and potassium iodide, is extremely slow, and slower when the experiment is performed with the eye in its normal condition than when the lens is placed in a test tube. Foreign substances which have been introduced into the blood are always demonstrable in the lens very much later than in the other parts of the body, if at all. Even after the introduction of such substances as potassium ferrocyanide and fluorescin directly into the eye, hours and even days may elapse before they appear in the lens.

The remarkable slowness of the process of diffusion into the lens must have a cause. The epithelium exercises a sort of physical selection. There is only a small amount of connecting material between the fibres of the lens, and this forms the primary path for diffusion in the organ. To this must be added the peculiar physical nature of the substance of the lens, a glutinous mass, which may be appreciated by crushing a lens between the fingers. This protoplasm evidently exerts a considerable resistance to the diffusion of soluble substances, especially when they are heterogenous, while the intercellular material, which forms the path for the nutrient substances to the cells, is fitted for greater diffusion.

One peculiarity of the lens substance, constantly met with in the pathology of the lens, as will be seen later in the origin of traumatic cataract, is its hygroscopicity, which is due to the large amount of albumin it contains. If a wound is made in the capsule of an animal's lens, and the latter is then placed in a 0.95% isotonic solution of salt, the lens substance about the wound becomes greatly swollen. Evidently two processes take part in the production of this swelling, osmosis between the contents of the fibres and the surrounding fluid, and a molecular imbibition of water by the lens fibres.

Important as osmosis and diffusion are in the nutritive processes in every cell, including the cells of the lens, we learn nothing more from them alone concerning these processes. The study of immunity, particularly through *Ehrlich's* theory of receptors, has revealed that the living protoplasm of the cells possesses many specific affinities, by means of which the cells appropriate and assimilate their nutriment. By these chemical groups, specifically arranged according to maximal affinity, each cell selects the substance that it needs. It can be demonstrated by a law which I discovered that these processes of cell nutrition apply also to the lens.

When the exchanges of substances between the blood serum and the lens are studied, it appears that not every antibody can enter the lens, *but only those to which it has a specific affinity.* By experiment it may be shown that the action of only one kind of hæmolysins is annulled by the reception of these cytotoxic bodies of the serum into the lens, while the other kind dissolves the blood cells, because the lens possesses no specific affinity to it. From these experiments it may be learned, and this is important with reference to the nutrition of the lens, that the appropriation of all such substances by the lens is subject to the law of the specific division of corresponding receptors. The more we study this subject the stronger becomes the evidence that the processes of pure osmosis, through which it was once hoped to penetrate the mysteries of cell metabolism, are insufficient to explain the appropriation of nutriment by the cells.

III. What Substances Are Appropriated by the Lens?

We know nothing whatever about what substances are appropriated by the lens from the intraocular fluid, or about the proportions these substances bear to each other, or about the time occupied by the process. Experiments with foreign substances can give us no information, because the lens does not live on such material. If such substances enter the lens and are harmless to the cells, they are excreted again without undergoing assimilation; if they are poisonous nothing can be inferred, concerning the normal nutrition of the organ, from the effects that they produce.

In view of the fact that the nutrition of the lens is subject to and provided for by diffusion and the specific affinities of the cell protoplasm the theory formerly held, that nutritive currents enter the lens in certain places, is untenable. Such a theory is not consonant with our present knowledge, is based on experiments with foreign substances which prove nothing, and no anatomical examination has revealed any preexisting tracts in the lens.

We know also that the nutrition of the lens and its transparency are independent of its connection with the zonula, because lenses remain clear for a long time after they have been dislocated into the vitreous. That such foreign substances as potassium ferrocyanide are usually met with in the neighborhood of the equator of the lens is explained by the fact that, after passing through the secretory organ, their concentration must be at its maximum for awhile at that point. Everyone conversant with the facts will therefore agree with *Leber* that the lens elements in need of nutrition draw their supply from all places where the nutriment has free access to the lens. But I believe, though *Leber* does not, that the anterior surface of the lens takes the essential part in the work of nutrition. *Hess* has shown that up to the time of birth the anterior capsule is covered by many vessels, and that such a vascular development indicates that the vessels are needed for the nutrition. It is not likely that the lens should suddenly lose at birth its ability to receive nourishment through the anterior capsule.

IV. The Constructive and Destructive Activity of the Protoplasm of the Lens

How the nutritive substances appropriated by the lens are used surpasses our knowledge. Every living cell is a chemical laboratory in which a constructive and destructive metamorphosis of the living protoplasm takes place, but we are unable to view the process in it, or in the lens.

The lens consists of about 65% water and 35% solids. Of this 35% rather more than 1% is composed of salts and such substances as cholesterin and lecithin, the remaining 33–34% of derived albumins. According to *Moerner* about one half of the derived albumins are soluble in dilute salt solution and resemble globulins. The insoluble derived albumins are called albuminoids.

The presence of chemically acting groups, called receptors, in this lens albumin is proved by the fact that solutions of the lens albumin are able to agglutinate strongly the blood corpuscles of rabbits, and these bodies act specifically only on rabbits' blood. The lens albumin is able also to fix tetanolysin, a toxin of tetanus bacilli, and so protect blood cells from this poison.

It can be shown also, by a series of experiments, that the lens albumin is able to fix certain hæmolysins in a specific manner. All these things go to show that the protoplasm of the lens has powers which can and do become active in the constructive and destructive metamorphosis of nutrient substances. Objections have been raised recently by *Salus* to the receptor nature of these constituents of the lens which are so efficient biologically. He believes their action to be that of lipoids, but the specificity of these actions is not to be explained by such a hypothesis. It is evident also that chemically efficient groups are present in the molecules of albumin in an organ 30% of which consists of albumin. That among them are groups which act as receptors is shown by the fact that specific antisera may be produced with lens albumin. This leads us to an important phenomenon, for the knowledge of which we have to thank *Uhlenhuth.*

In a serum productive of specific precipitates, e.g., one that permits the recognition of human blood and organic albumin alone, distinct precipitates are formed by the admixture of the thinnest dilutions of human blood. This phenomenon is of great forensic importance. But no precipitates are formed in the serum by solutions of the human lens, which proves that the derived albumin of the lens differs biologically from all other albumins in the human body. Inversely: A serum prepared through previous treatment of an animal with albumin from the lens of an ox, for example, will not only precipitate the albumin from the lens of an ox, but likewise solutions of albumin from lenses of all kinds of mammals, while the albumin of the blood does not react with such a serum. In this way the blood of different animals can be distinctly recognized, but the albumin of the lens of an ox cannot be distinguished from that of a human lens.

Identical derived albumins are contained in the lenses of all mammals. The fact that the structure of its albumin is primitive and common to different species of animals leads us to suppose that the lens is an organ which is formed at an early period of embryonal life, before the advent of the time for a further transformation of its albumin to correspond with the species.

To recapitulate we may say that the lens obtains its nourishment through osmosis with the intraocular fluid and through the specific affinities of its protoplasm. Disturbances in this process of nutrition lead us into the pathology of the lens, to which we will now turn.

52. Visual Disturbances Due to Changes in the Lens without True Cataract

I. Cataracta Nigra

We have here an aged patient who says that he can no longer see. Such a condition may be due to many causes, for the weakness of old age may give rise to various morbid changes, and our first duty, when a patient complains of inability to see, is to examine the refractive media for the cause.

In this patient the cornea and aqueous are clear, but an almost dark brown reflex is obtained from the pupil by oblique illumination, especially when the pupil is dilated. There is no doubt that this reflex comes from the lens and is greatest in its centre, while toward the periphery the yellow, or brown color passes over into a diffuse, grayish reflex. When the age of the patient is taken into consideration this appearance suggests cataract at once, but an examination with the ophthalmoscope shows the lens to be so transparent that the fundus can still be made out.

There is in this case no true opacity of the lens, such as is understood to be meant by the word cataract. The Arabs called the opaque lens ''the water that falls in the eye''; hence the name that has been handed down from the Middle Ages, cataract, waterfall. Under this term, cataract, is comprehended all the diseases of the lens which are associated with visible opacities. No such opacity is present in this case, and yet the patient complains of visual troubles which may be due to changes in the lens. Central vision is found, on testing, to be greatly reduced, and even deep blue appears gray to the patient.

This case is a typical example of the fact that the physiological changes in the lens due to age may exceed their normal limits and lead imperceptibly into pathology. The lens undergoes a process of sclerosis which produces a yellow discoloration in its nucleus; when this sclerosis is excessive the yellowish becomes a brown discoloration and gives rise to such disturbances of vision as those complained of by this patient. Such a lenticular change is called cataracta nigra, although no true cataractous opacities are present.

Examination of the lenticular reflexes shows this to be a case of great sclerosis of the lens. The anterior reflex of the nucleus is yellowish in cataracta nigra and the posterior reflex from the cortex, which is scarcely visible, may appear brownish.

The explanation of the interference with vision is that the brown discoloration of the lens causes a considerable absorption of the light, while at the same time the increase in the sclerosis of the nucleus results in reflection of much of the light, as may be seen from the strong reflex from the lens, and it happens not infrequently that the brown discoloration is associated with a diffuse cloudiness of the nucleus, such as is met with in nuclear cataract. In my opinion cataracta nigra differs from the normal senile lens only in the high degree of its sclerosis.

The idea has been advanced repeatedly that the brown discoloration comes from the coloring matter of the blood in the form of hæmatin, or melanin, but this theory is not tenable. Like *Hess*, I have been unable to find spectroscopically any signs of a derivate of the coloring matter of the blood. It has also been claimed that the black color of cataracta nigra is produced by pigment granules from destroyed pigment epithelium cells, but this also is incorrect, because such substances cannot pass through the intact lens capsule.

Vision can be improved in these cases only by the removal in toto of the lenses. They may be very large, as they consist only of large nuclei. The chances of the operation are very good.

Under the microscope the entire lens up to the capsule is seen to be transformed into a nuclear mass. The epithelium of the anterior capsule is very small and the nucleus contains very few cells and nuclei as compared with a younger lens.

II. Lens with Double Focus

This patient, who is in his forty eighth year, states that during the past year he has become very nearsighted, though he previously could see well at a distance, and has never worn, or needed a glass for nearsightedness.

Myopia develops in youth and the normal eye begins to become presbyopic about the forty fifth year, so a history of development of myopia in advanced life immediately suggests two possibilities.

We not infrequently meet with people who are happy that they can read once more without their presbyopic glasses. The physician knows that such a myopia, appearing in an elderly person, is often a sign that a cataract is being developed, but the myopia can occur without the formation of a cataract.

When the pupils of such a patient have been dilated it can be seen by oblique examination that the central portion of each is slightly gray, or yellowish gray. The phenomena seen with the ophthalmoscope are much more characteristic.

First it must be ascertained that the lens is perfectly transparent and contains no opacities, which appear dark in an ophthalmoscopic examination. Then, when the mirror is rotated slightly from side to side, as in a skiascopic examination, a most striking phenomenon is to be seen. The central part of the pupil appears somewhat darker and distinctly separate from the brighter peripheral portion, in spite of its transparency, and with each movement of the mirror a circular shadow comes and goes between them.

Still more remarkable is the difference in the refraction between the central and the peripheral portions of the pupil; the central field moves in the same direction as the movement of the concave mirror, while the movement of the peripheral zone is in the opposite direction. In other words: there is a fairly high degree of myopia in the central part of the pupil, with emmetropia, or perhaps hypermetropia, in the peripheral part. Now it is clear why the patient said that he had become nearsighted.

Subjective tests show that the patient brings a book very close to his eyes, and that a strong concave lens is necessary to enable him to see at a distance.

Two refractive conditions are present in the one eye. This has been caused by a change in the lens and therefore the clinical picture has received the name of "lens with double focus." It has also been termed false lenticonus and pseudocataract (*Halben*). It furnishes another example of how changes in the lens may cause disturbances of vision without the presence of cataract.

The origin of this abnormally great difference in the refraction of the central and peripheral portions of the lens may be studied by means of the lenticular reflexes. When the reflex from the nucleus is compared with that obtained in another person of the same age with a normal lens it is seen to have an increased light strength. The greater the difference between the index of the cortex and the index of the nucleus the more distinct becomes the reflex from the nucleus. Hence this anomaly depends upon an abnormally great difference between the indices of the cortex and of the nucleus.

This is indicated also by the great difference in refraction between the central and peripheral portions of the lens. The question now arises: What is the cause of this abnormally great difference between the index of the cortex and that of the nucleus? *Hess* has shown that under normal conditions the transition from the index of the cortex to that of the nucleus is not gradual, but sudden. There are a priori two possibilities: Either the cortical index is abnormally reduced by diffusion in commencing cataract, as thought by *Demicheri*, or the index of the nucleus is raised by an abnormal loss of water, as suggested by *Guttmann.*

The theory that the cause of this anomaly of a lens with a double focus is an abnormal increase of the index of the nucleus seems the more probable because it is not always a forerunner of a true cataract. *Hess* has called attention to the fact that perhaps a greater curvature of the surface of the nucleus needs to be taken into consideration, because the greater the curvature of the surface of the nucleus the greater is the refraction of the rays of light when the difference between the indices is the same. However this may be, the fact remains that such changes in the lens without true cataract can produce important disturbances of vision. In such cases we try first to improve the vision with glasses, but if the visual disturbance is so great, and the refraction is so difficult to correct that this cannot be done, no remedy remains except the extraction of the lens.

53. The Most Frequent Form of Senile Cataract, Cataracta Senilis Subcapsularis

True *opacities* in the lens of various forms and extent are met with in a great many elderly and very old people. Every layman knows that a cataract which appears in old age is called a senile cataract. This is a diagnosis, but one that hardly suffices for the general practitioner and surely does not suffice for the ophthalmologist, for quite different diseases of the lens have been grouped together under the term senile cataract.

One form we have studied already, the cataracta nigra. It is a question whether this is a real cataract, because the lens is only abnormally brown and does not contain any true cataractous opacities, although the disturbance of vision produced by the abnormal sclerosis may be so great as to necessitate extraction of the lens.

The conditions are quite different in the other cataracts which are met with in old age. In all of them actual opacities, manifold in form and extent, may be seen in the lens. The forms met with late in life are: 1, subcapsular cortical cataract; 2, supranuclear cataract; 3, nuclear cataract; 4, punctate cataract. The differentiation of these forms is not purely of theoretical interest, as will soon appear, and whoever would determine the particular form of cataract in an individual case must not only be acquainted with, but use in every case the methods of clinical examination at our disposal. The exact diagnosis of the form of a senile cataract is possible only when the pupil is dilated and these four methods of examination are employed one after the other: 1, oblique illumination; 2, simple illumination of the pupil with the ophthalmoscope; 3, careful examination of the cataract through a strong plus lens attached to the ophthalmoscope; 4, examination with *Zeiss*' loupe.

The Subcapsular Cortical Cataract

For the commonest form of senile cataract we have adopted the name given it by *Hess*, subcapsular cortical cataract, to impress the fact that the morbid changes take place in the outermost cortical layers of the lens. The lenticular fibres which are situated just beneath the capsule are the ones which become opaque and finally disintegrate. The nucleus remains wholly unaffected, except in a few cases in which it becomes smaller. The general practitioner should be well acquainted with this form of cataract, for it is this chiefly which is met with in patients who seek him for advice. He may be able to conjecture the presence of an incipient senile cataract from the troubles of the patient, but he should be able to confirm his suspicions objectively to make his diagnosis.

The symptoms complained of by the patients vary exceedingly. One would think that failure of vision would usually first lead the patient to the physician, but some have very vague complaints. They may complain of muscæ volitantes, and then it may be asked whether the muscæ remain fixed when the eye is quiet, or keep on moving and sink. In the latter case they may be due to floating opacities in a fluid vitreous, but when they are caused by lenticular opacities they cannot change their places.

Some patients complain that they seem to be seeing through a veil, sometimes that they have double, or quadruple vision. In such patients one eye may be closed to ascertain whether they still have diplopia. When we come to the study of muscular paralyses we will find that diplopia due to paralysis disappears at once when one eye is excluded from participation in the act of vision. If the diplopia is due to a lenticular opacity in one eye it must still be present when the other eye is closed.

Polyopia is still more interesting. *Fuchs* tells in his textbook of a lamplighter in a castle who saw thousands of lights because of his polyopia, and believed that he was dealing with a ghost. Diplopia and polyopia are produced by irregular astigmatism of the lens, and it may be imagined what optic irregularities may take place in such an organ when it is undergoing disintegration. It has been mentioned that myopia may appear in the development of senile cataract, but in the majority of cases the complaints of patients with incipient cataract are very vague. Therefore it is important for the physician to know that there is scarcely a disease of the eye which can be recognized so positively and with so little trouble as incipient cortical cataract. This can be shown by means of several patients who exhibit the primary stage of this disease.

The pupil is first dilated and then by *focal illumination* divergent, gray and gray white, wedge shaped opacities can be seen beneath the capsule in the periphery of the lens. Their apices are directed toward the anterior pole, and the inner and lower margins are particularly affected. The pupillary area is still clear, at least to the naked eye.

Before the pupil is dilated its area appears gray on ordinary examination, but this does not necessarily indicate opacity. When it has been dilated such opacities as those just described are found in the peripheral portion of the lens behind the iris. This proves that the artificial dilatation of the pupil is of great importance in the diagnosis of an incipient senile cataract, and also that the subcapsular cortical cataract appears to start by choice in the region of the equator of the lens.

Examination with the ophthalmoscope reveals two new points. First, all of the lenticular opacities, which appeared to be gray, or gray white by focal illumination, now stand out as dark, or black against the red background of the rest of the pupil, for all opacities of the refractive media of the eye appear to be dark in the light transmitted by the ophthalmoscope. Second, more opacities come into view than by focal illumination, and the very first signs of senile cataract may be seen.

The lens cells and fibres do not disintegrate and become opaque evenly, but frequently radii as bright as glass, which have been compared to cracks in clear ice, appear between the breaking down lens fibres and form the first indication. At first they can be perceived only by alternating movements of the mirror. There is no doubt that they are caused by accumulations of water between the fibres. The constituents of the lens fibres undergoing disintegration gradually make them opaque, and finally, together with the opaque lens fibres in their neighborhood, they produce the clinical picture of cataractous opacities, and in this simple way the exact position of each may be ascertained. In advanced cases scalelike opacities in the posterior layer of the cortex may be recognized by oblique illumination, but in the primary stage their positions can be learned only by means of transmitted light. If such an eye turns upward while undergoing examination with the ophthalmoscope a part of the lenticular opacities appear to move downward. This is parallactic displacement. The apparent change of place of part of the lenticular opacities in the opposite direction to the movement of the eye shows that they are in the posterior corticalis. This exact determination of the position and extent of the cataractous opacities is of importance to the ophthalmologist if he would watch their advance. The diagnosis of subcapsular cortical cataract is established by means of focal illumination and ophthalmoscopic examination, together with the simultaneous observation of the parallactic displacement. These methods can be refined by the aid of an additional strong magnifying glass.

When light is focussed upon the eye, and the eye itself is observed through a second magnifying lens, many details are perceived, especially of the opacities in the anterior cortex, which would otherwise escape notice. We use for this purpose, here in the clinic, *Crapski's* binocular microscope, by means of which we obtain not only magnification, but also binocular stereoscopic vision. No case of cataract goes through our clinic without an examination with this instrument.

Various convex lenses may be interposed in the ophthalmoscope, by means of the rotating disc, so as to permit the opacities to be studied under different degrees of enlargement, and it is astonishing how much more can be learned about them in this way.

In another case of subcapsular cortical cataract single fine, dark gray lines may be seen in the lens by focal illumination, but with the ophthalmoscope an entire series of clear, cleftlike formations, arranged in a radiating form, can be perceived in the equatorial region. By moving the mirror to and fro part of the clear radii almost completely disappear, to come again into view in other positions of the instrument. At times they appear bright, at others they cast dark shadows. The longer the examination, the more shadows appear. This proves that these clear radii are situated just beneath the capsule and not in the deeper layers of the cortex. Such very minute opacities of the lens are perceptible only by means of the mirror, either alone or with a magnifying lens.

It is important that the ophthalmologist should record graphically on a chart the extent of the morbid process found in the lens by means of this examination with the ophthalmoscope. Each examination to be recorded must be made with a magnifying lens of a certain strength and with the eye looking directly forward. In this chart the visible field of the dilated pupil is divided into sixteen sectors, and also into a peripheral *a*-zone, a middle *b*-zone and a central *c*-zone. By this means the presence and position of each lenticular opacity can be recorded very accurately. The differentiation of anterior and posterior cortical opacities may be indicated by the use of different colors.

We have thus far been studying the initial stage of subcapsular cortical cataract, which passes through several stages of development. It evidently starts from an increased permeability to water on the part of the external cell elements of the lens. Accordingly, in one patient a number of clear spaces can be seen in its outermost periphery, which were caused by fluid between the lens fibres in the equatorial region. *Hess* has also called attention to minute, roundish, vesicular vacuoles, which may be perceived by careful examination here and there beneath the epithelium of the anterior capsule in the pupillary region. These also indicate an impairment of the regulation of the amount of water in the lens by the epithelium. The number and extent of the clear spaces beneath the capsule gradually decrease. Their contents begin to become cloudy, and then quite a number of gray, or gray white, opacities appear: radiating opacities have developed from the waterlike radii.

It is improbable that all the opacities originate in this manner. *According to my observation they are produced rather by a direct disintegration of the lens fibres.* It must not be forgotten that, throughout the entire process of destruction of the lens elements, an exchange takes place between the contents of the fibres and the fluid that has entered, and that this varies according to the depth and lateral extent, as well as the age of the lens. The clinical picture of an incipient subcapsular cortical cataract therefore varies exceedingly. We meet with irregular gray patches in addition to the regular radiating figures, but in the incipient stage small sections of the lens always have been affected first by the morbid process, and often these can be discovered only when the pupil is dilated. The eye is otherwise normal and usually the vision is impaired slightly, or not at all, even when the opaque sectors encroach upon the pupillary area. It happens not rarely that the involvement of the posterior cortex, particularly in the posterior polar region, is responsible for the failure of vision.

In this stage of incipient cataract the condition of the vitreous should be carefully noted. Anyone who is accustomed to do this is able to confirm the statement that scarcely a case of incipient cortical cataract is met with in which opacities cannot be distinctly recognized in the vitreous. The electric ophthalmoscope is of great service for this purpose; it is surprising how much more can be perceived with it in this examination. The fact that the fluid of the vitreous is found very often to be not normal seems to me to be of great interest in the pathogenesis of diseases of the lens.

The next case presents quite a different picture. The opacities have increased in size and number, the disintegration of the fibres is further advanced, and we meet with entire strata that are opaque. The apices of the sectors come partly into view even when the pupil is not dilated. The radiating arrangement is still distinct, and the posterior can still be distinguished from the anterior opacities in the cortex. The red reflex can still be obtained with the ophthalmoscope, but the dark portions are much more massive. The anterior chamber at this time is distinctly shallower than it should be, as may be seen by comparing the two eyes, if the other one is normal. This is due to the swollen condition of the lens. The studies which have been made hitherto concerning the proportionate weight of the opaque human lens are unsatisfactory, for it is not easy to obtain unobjectionable material for the necessary control lenses.

In still another case the area of the pupil is uniformly gray, and a glance with the ophthalmoscope shows that it is no longer possible to see into the eye. The cortical layers beneath the capsule are completely opaque. A sectorlike demarcation of the swollen masses of fibres is perceptible still in the pupillary area, especially when the pupil is dilated, and the stellate rays are frequently distinct.

The cataract in the other eye is not yet ripe. This may be shown by allowing light to fall obliquely upon the eye so that the margin of the pupil casts a narrow, dark shadow. Great clinical importance was formerly ascribed to this shadow of the iris. This shadow is formed on the side of the pupillary margin turned toward the light, because the rays of light pass through a transparent layer of cortex before they are intercepted by an opaque layer that is situated more deeply. Hence its presence proves that the cataract is not yet mature. At this time the anterior chamber is no longer shallow, so the size of the lens must have become reduced by loss of water and inspissation of the opaque masses.

If a cortical cataract is not removed by operation after it has advanced thus far, further changes take place in the lens. We have here a patient with a very deep anterior chamber and a tremulous lens, which is evidently shrunken. Attention is attracted at once to the peculiar white dots and spots which lie just beneath the capsule in the pupillary area. Beneath them can be seen the brown nucleus of the lens. This is the stage of cataracta hypermatura. The size of the reflex from the nucleus is to be noted, because it will take part in determining the size of the section to be made later at the operation. This is not the only picture we meet with in hypermature cataract. Instead of having formed a mass containing little water the cataract may be transformed into a milky fluid, and the lens is uniformly bluish, with no traces left of its stellate appearance. The brown nucleus has sunk to the bottom of the sac formed by the capsule, in which it can be made to move about by changes in the position of the head. Such cataracts are named fluid, or Morgagnian cataracts. The fact that the lens is tremulous in this stage proves that its suspensory ligament must be relaxed.

The course of a subcapsular cortical cataract represents a death of the lens until finally it lies a dead organ within the human eye.

The clinical picture cannot be understood properly without a study of its microscopic anatomy. Simple as the construction of the lens appears, the details are manifold in the pictures met with in the histological examination of the diseased organ. Two phenomena are constantly met with: 1, degeneration taking place in the epithelium of the capsule with active proliferation; 2, signs of disintegration preponderating in the lens fibres themselves. The changes that take place in the epithelium of the capsule are investigated easily, because cataract patients continually furnish fresh material.

On one slide it can be seen that the connection between the epithelium and the capsule is relaxed, numerous cells are deficient, here and there only remains of nuclei may still be seen, and over whole areas the cell protoplasm appears to be greatly degenerated.

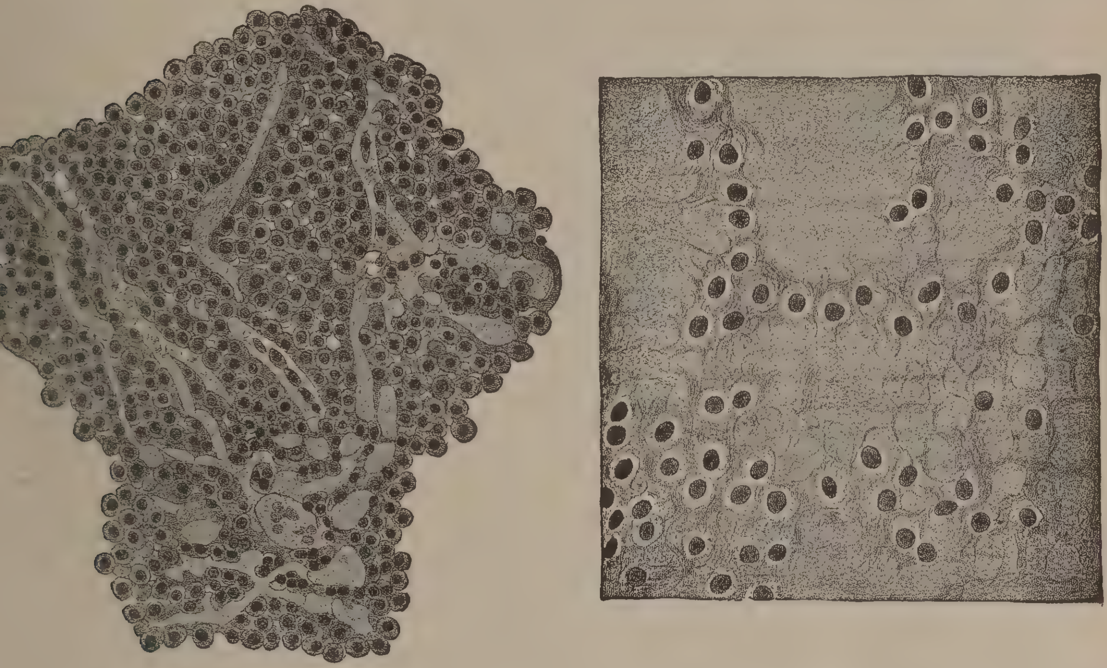

FIG. 33.—Degeneration of the Epithelium of the Capsule in Senile Cataract, after *Hess.*

On another slide may be seen irregular striæ in which cells are wanting here and there. Numerous vacuoles lie between the cells that are preserved. In other places normal, sharply defined cells alternate with badly stained ones which have ill defined margins. Proliferation of the epithelium can be seen distinctly about such zones of degeneration. It seems as if the lens had tried for a long time to resist the harmful influences which result in this form of cataract. On another slide morbid changes in the cell nuclei can be demonstrated. The disintegrating nuclei are surrounded by numerous minute vacuoles and are associated with many other changes. Sometimes an epithelial coat, composed of irregular cells of various shapes which come from the cell elements of the equator of the lens, is found on the posterior capsule. One of the commonest forms of degeneration of the epithelium of the capsule is the so called vesicle cell (Blæschenzell). One of

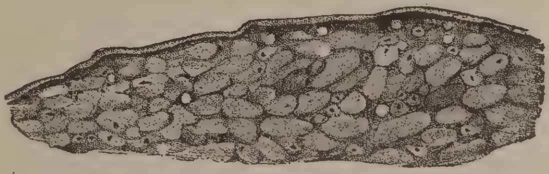

FIG. 34.—Pseudo Epithelial Layer on the Posterior Capsule.

the first morbid symptoms in the lens fibres is the swelling, and from these swollen fibres vesicle cells may be formed. From the diseased fibres flaky masses enter the fluid collected between them, and finally they degenerate into a flaky liquid. Here and there remains of fibres are met with, having proliferated epithelial cells about them. Usually the nucleus of the lens is differentiated quite sharply from this zone of disintegration, and its compressed fibres appear to be normal. More rarely its surface is corroded and presents irregular defects. In the hypermature cataract the disintegration of the cortex is complete, and even the epithelium of the capsule may be nearly or quite destroyed.

54. Various Other Forms of Senile Cataract

Cataracta Nuclearis

In subcapsular cortical cataract the morbid process takes place in the outermost layers of the cortex, while the nucleus escapes injury, but we meet with other forms of cataract, one of which is exemplified by this patient, in whom the nuclear portion of the lens appears to be uniformly and diffusely opaque, while the cortical portion is perfectly clear and transparent. No clefts, radii, or radiating opacities are present, and the only thing that can be positively determined is that the nuclear opacity is without a sharp line of demarcation, but blends gradually with the transparent cortex. This is known as the intranuclear senile cataract. This phenomenon follows no fixed law. Cases are met with in which this nuclear opacity is associated with subcapsular cortical cataract. I am convinced that in such cases two entirely different morbid processes occur together, that the lens is attacked by two different diseases either simultaneously, or in succession.

This form of cataract is much less common than the ordinary cortical variety and is met with in younger people. Nuclear cataract usually develops about the age of forty. At that time the nuclear opacity is small and faint. It is much less distinctly visible with the ophthalmoscope than by oblique illumination, and reminds one of abnormal physiological sclerosis of the nucleus without true opacity. In the course of years the intranuclear opacity increases gradually until single punctate spots can be perceived within the nucleus on careful examination. In other respects little concerning its nature has been learned from clinical observation and histological investigation. The lenticular reflexes are just the same as in other persons of the same age with healthy eyes.

Scarcely any characteristic changes have been found in sections of lenses with nuclear cataract. In one lens the innermost portion of the nucleus appears to be uniformly homogeneous, so that the individual lens fibres can no longer be distinguished, but in another case this is less marked, and such differences are met with even in quite normal old lenses. There is frequently a dry appearance of the nuclear portion, which stands out sharply from the lustrous cortex, in the freshly cut lens with a nuclear cataract.

Cataracta Supranuclearis

Still more rare than the nuclear variety is the form met with in older persons and known as supranuclear cataract. In this form the opacity is situated not in the outermost portion of the cortex and not within the nucleus, but where a line of demarcation separates the surface of the nucleus from the cortex. Under normal conditions the transition from nucleus to cortex is not uniform, as *Hess* has shown, and it is only when a true opacity is present that we can speak of a supranuclear cataract. When studied with *Zeiss*' loupe such opacities have a markedly whitish color and their position between the nucleus and the cortex can be made out distinctly.

Hess found, by anatomical examination, a longitudinal hollow space filled with homogeneous masses extending from the equator of the nucleus into the supranuclear zone, which was sharply differentiated from the nuclear zone. Near it lay other, irregularly defined, smaller patches. All of these areas of degeneration decreased toward the nucleus, the centre of which was free from such morbid changes.

A final form of cataract met with in old persons is that known as cataracta punctata cœrulea. Properly speaking this should not be included among the forms of senile cataract.

Cataracta Punctata

The incipiency of this form of cataract usually reaches back to a younger period of life, not rarely to the twenties, and some authors designate it as a form of congenital cataract. It is of great interest to us because of its being, in my experience, next to subcapsular cortical cataract, the form most frequently met with in old people, and because it is very apt to be mistaken for ordinary senile cataract when an insufficient examination has been made. This mistake does not happen when met with in young, or middle aged patients. It is by no means rare in this part of the world, but forms nearly half of all the cataracts we meet with in old age.

A characteristic feature of this form of cataract is the great difference between what is seen with the ophthalmoscope and the condition revealed with *Zeiss*' loupe; so much less can be seen with the ophthalmoscope that in many cases the positive diagnosis of cataracta punctata can be made only by means of the latter instrument, which reveals roundish, sharply defined clouds that have a peculiar bluish lustre in the supranuclear layers. The first signs may be met with between the ages of twenty and thirty. When the cataract is more developed numerous bluish opacities may be seen in the anterior portion of the lens, ellipsoid in form, arranged like palisades, the axes of the ellipsoids forming radii. They may extend almost into the pupillary region, although in the initial stage they lie hidden behind the iris and can be discovered only when the pupil is dilated. A more precise examination reveals about and between these palisades an innumerable quantity of very minute, round points, which frequently appear like dust in the lens on examination with the ophthalmoscope. Another characteristic feature is that the palisades do not start from the equator, like the opacities of cortical cataract, but from a preequatorial zone, so that the immediate neighborhood of the equator is found to be perfectly free. Another feature is that the anterior and posterior poles are the parts longest exempt from this degeneration in the great majority of cases. This circumstance and the fact that the opacities are so sharply limited explain why vision remains so good in this form of cataract, even in its final stages. Vision fails only when the ellipsoids have become large and acuminate opacities have been added, which extend to the anterior and posterior poles of the lens. Decades may elapse before this takes place. Entirely different laws underlie the development and extension of this form of cataract from those of the subcapsular cortical variety. Whoever knows this can give his patients a better prognosis concerning the advance of the cataract.

Caractata punctata is by no means always stationary, but it develops much more slowly than the average cortical cataract. The positive diagnosis of cataracta punctata is of interest from the point of view of the operation, because experience teaches that it can easily be removed in toto and is particularly fitted for the extraction with round pupil. Very often a lens with cataracta punctata is attacked by subcapsular cortical cataract, which seems to cause a sudden advance of the former disease. The gray, or rather, dirty gray brown, indistinct subcapsular opacities may be distinguished by means of *Zeiss'* loupe from the bluish, sharply defined, and more deeply situated opacities of the punctate cataract. The prognosis in such mixed cases is different, although the progress seems to be slower than it is in most cases of purely cortical cataract.

In a section of a punctate cataract homogeneous, and in part finely granular masses may be seen to lie between the apparently normal cortical fibres. In places the subcapsular layers of the lens present spindle shaped spaces between the fibres, some of which are filled with finely granular masses. The cause and the nature of this form of cataract are wholly unknown.

55. Genesis of Senile Cataract

We cannot pass by the subject of senile cataract, which yearly blinds many thousands of human eyes, without a consideration of its pathogenesis. It has been known from the earliest days of medical tradition, and yet the riddle of its causation has not been solved.

Is it a true disease? Or is it a physiological necessity that the lens should begin to become opaque when the man has reached a certain age? When the silver threads appear in the hair, when the body bends under the weight of years, when life with all its sufferings has consumed decades, does Nature render it needful that our lenses should live no longer, that cataract accompanies, or starts the decay of the organism? There are yet to day those who believe that every man will have a senile cataract finally, if he lives long enough. Such an idea is supported by the fact that lenticular opacities are to be met with in old people very frequently, but this is not cogent proof that senile cataract is a physiological necessity. Even though many old eyes have lenticular opacities, yet it remains a fact that only a small fraction of the people who grow old develop senile cataract. As long as the other degenerative changes which take place in the organism in old age are not considered to be normal, and as long as there is scientific justification for the recognition of different forms of senile cataract, we must not consider it to be a physiological involution of an aged organ, but a disease of the lens.

Concerning the genesis of the rarer forms of cataract met with in old age we know nothing, but that of the commonest form, the subcapsular cortical cataract, has long been a subject of study. I will not weary you with ancient theories, but turn at once to the time when ophthalmology was placed on the basis of modern physical science and exact experimentation.

The theories that have been advanced to explain the occurrence of senile cataract may be divided into three categories.

First come those in which the *cause of senile cataract is sought for in the lens itself*. The first hypothesis elaborated concerning the origin of senile cataract was that defended by *Otto Becker* in 1876, of an irregular sclerosis of the lens. This theory has prevailed even to the present time. *Becker* says: "It has to be assumed in senile cataract that the sclerosis of the lenticular nucleus, when not perfectly constant and uniform, so as to lead to the formation of a cataracta nigra, interferes with the uniform distribution of the nutritive fluid and thus gives rise to a relaxation of the layer adjoining the nucleus. This relaxation has also a mechanical basis. When the nucleus becomes condensed and reduced to a small volume it must exert a tension upon the more peripheral parts. The cortical layers beneath the capsule about the anterior and posterior poles may yield readily, because the capsule is not fixed at those points, but the condition is different in the region of the equator. In the first place the coherence of the cortical fibres to each other is less because the youngest lens fibres are to be found at this place. At the same time the zonule of *Zinn* fixes the capsule externally, so as to prevent the withdrawal of the equatorial part of the lens from the ciliary body and its approximation toward the axis of the eye. Hence it cannot be surprising that in senile cataract there is first a loosening of connection, a separation from each other of the cortical layers, and then an opacity of the lens fibres, a molecular disintegration, a formation of cataract."

The questions immediately arise: Why does this abnormal sclerosis happen in only a part of the lenses that are growing old? And why does this process take an abnormal course? *Becker's* theory can give no satisfactory answer to these questions. He only assumes that the nucleus of the cataractous lens may be qualitatively different from that of the healthy lens, that in this anomalous chemical behavior of the nucleus may be sought the cause of its greater contraction, and that perhaps by this chemical difference it may be determined in the forties whether a man will have a cataract in old age. But this chemical difference between the nucleus of the cataractous and of the healthy lens has not been proven, and thus the foundation for such a theory of predestination is lacking. In spite of its evident weaknesses *Becker's* theory enjoys a high regard, has preserved its authority up to the present day, and has been adhered to by *Deutschmann*, who says: "The beginning of the senile cataract is to be sought in a disintegration of lens elements within the intact senile lens. The true basis of this disintegration lies in an irregular process of sclerosis. The water, which the lenticular nucleus gives up in its sclerosis and is harmless to a regularly and uniformly sclerosing lens, clouds the lens undergoing this faulty and irregular process by swelling its fibre elements. To the disintegration of fibres thus started succeeds a lively diffusion between the destroyed lens elements and the surrounding media, especially the aqueous."

Recently *Hess* has raised serious objections to *Becker's* theory. *Becker* had referred in part to *Priestley Smith*, who had found that lenses with commencing opacities were frequently somewhat lighter and smaller than clear lenses of the same age, and *Becker* had taken this to be proof of a greater contraction of the nucleus in the cataractous lens. *Hess* properly maintains that a frequent, but by no means regular, decrease in volume and weight cannot be referred offhand to a contraction of the nucleus. The number of lenses that have been weighed is much too small, the fluctuations found in weight and volume are much too great, to permit of the conclusion that a contraction, a shrinkage, of the nucleus had taken place. Moreover it must not be forgotten that it is clinically demonstrable that in the first stage of subcapsular cortical cataract the lens distinctly increases in volume. For these reasons we have to drop *Becker's* hypothesis.

Among other theories that have been promulgated is that of *Schoen*, who likewise located the cause of senile cataract in the lens itself, though differently. He believes that the cause of senile cataract is the accommodative effort. *Schoen* says: "Continuous hard pulling on the fibres of the zonule produces detachment of the capsule in little folds, proliferation of the epithelium of the capsule in a little seam behind the equator, corresponding to the insertions of the middle fibres of the zonule, and in a second seam in front of the equator which corresponds to the insertion of the anterior fibres. Later the epithelial cells change into vesicular cells and become forced into the cortex of the lens, the capsule separates from the lens at the equator, and now disturbances of nutrition are created which lead to the further development of a cataract.'' According to such a theory lenticular opacities would be expected much sooner, in the younger years of life, when the mechanism of accommodation is most used, rather than in old age, when the accommodative changes in the form of the lens are very much less, or nonexistent. This hypothesis has not been accepted widely. It is untenable because, according to *Hess*, the presumptions of *Schoen* concerning the process of accommodation are incorrect, and also because clinical experience does not show that hypermetropes are more liable to senile cataract, or that cataract develops in them earlier in life. The same reasoning applies, according to *Hess*, to the theory advanced by *Schoen* and others that astigmatism takes part in the causation of senile cataract.

The cause of the development of senile cataract has been sought in the lens in still another way. *Magnus* believes that the hardening of the lens interferes with the currents of nutritive fluid, that a stasis of the latter occurs in certain regions of the lens, and that in old age the power of resistance of the lens fibre to such stases is reduced. But it is known that there are no nutritive currents in preexisting paths in the lens, and this disposes of this theory, entirely aside from the fact that it leaves unexplained the ultimate cause of such disturbances of the currents.

Finally *Moerner* says in his studies concerning the derived albumins of the lens: ''Possibly cataract is to be looked upon as the final consequence of this advancing albuminoid transformation of the lens fibres, in that after loss of the soluble derived albumins the lens can no longer transmit light satisfactorily.'' This chemical hypothesis does not hold good for numerous reasons. The physician would learn by what processes in the organism of the patient the disintegration of the lens fibres is caused. None of the hypotheses in which the ultimate cause of senile cataract is placed in the lens itself, and only in the lens, is able to explain the clinical picture of subcapsular cortical cataract.

Next we come to a group of ideas that form a hypothesis which may be called the chemico physical theory. It rests upon the studies of *Peters* and his pupils, and is characterized by the fact that it lays much more weight upon the osmosis of the lens with the fluid that surrounds it, and thus extends its sphere of activity outside the lens. *Peters* believes that constant differences of pressure must exist between the contents of the anterior chamber and of the lens to promote the normal nutrition of the latter. There must be, he thinks, a higher molecular concentration in the lens than in the anterior chamber, and that the maintenance of this is a duty of the epithelium of the capsule. Any increase of the molecular concentration of the aqueous would be critical to the lens, because then the osmotic exchange, and so its normal nutrition, would be disturbed.

Now it may be possible that, as a result of senile changes in the secretory organ, the aqueous may contain an increased quantity of salt. He considers it probable ''that a trifling increase in the concentration of the aqueous is of great importance, in so far as it is able to call forth no great diminution of the intracapsular pressure, but a certain degree of osmotic equilibrium between the lens and the aqueous, and so may result in at least a diminution of the currents through the lens. Thereby the central part of the lens will have to suffer, and so it can be readily imagined that a condensation takes place there, while the epithelium and cortex continue to be nourished normally. The lens responds to the total inhibition of the currents not only by a condensation of its central part, but also by a nutritive disturbance in its cortical layers, to which is to be added the diminution of pressure within the sac of the lens as an essential factor." A fact of osmosis contradicts this hypothesis. Numberless milliards of red blood corpuscles obtain nutrition although not surrounded by an anisotonic solution in the blood. On the contrary blood serum is considered the best isotonic solution for the blood cells. It is therefore questionable whether the case is essentially different with the lens, whether osmotic differences of pressure must be constantly present between the aqueous and the contents of the lens. Doubt in regard to this is justifiable. The statements of some authors, that the osmotic pressure of the aqueous may be higher than that of the blood, may indicate that an increase of the molecular concentration of salt in the aqueous is actually welcome to the lens.

But it has been shown that the osmotic pressure of the intraocular fluids is not higher than that of the blood, that the physiological fluctuations of the osmotic pressure in the blood serum are constantly transmitted to the intraocular fluids, and that a continual mutation takes place to which the lens is adapted just as well as the blood cells. From the results of the study of osmosis I believe it to be impossible for an abnormally high molecular concentration to be maintained in the aqueous. Even if an increased amount of salt appears in the aqueous as the result of senile changes in the secretory organ, the osmotic equilibrium must be restored immediately by osmosis with the fluid of the vitreous and with the contents of the surrounding bloodvessels. Large quantities of salt introduced into the anterior chamber of an animal are resorbed immediately, and the assumption that a transient increase of the concentration of salt is harmful to the lens is contradicted by the results of this injection of salt into the anterior chamber.

In addition to this it has not been proved that the development of a senile cataract begins with a shrinkage of the nucleus. If *Salffner's* findings in the experimental production of naphthalin cataract are taken in connection with the results of clinical observation, it must be admitted that the first symptom of subcapsular cortical cataract that can be perceived is not a shrinkage of the nucleus, but an increased admission of water into the cortical layer. When finally it is considered that the concentration of salt is but a single factor in the nutrition of the lens, and that the relations of the living cell elements to the other nutrient materials are disregarded, it will be agreed that the hypothesis of *Peters* is too simple to deal with the conditions in the development of senile cataract, which are evidently very complicated.

The third and final group of theories deals with the relations that exist between the entire organism and senile cataract. In view of the fact that as a rule senile cataract affects both eyes, though not uniformly, it could not fail that the ophthalmologist should hold the entire organism responsible for this phenomenon. Such an idea may be traced back to the earliest days of medical knowledge. I cannot enumerate all the theories formed in ancient times, in the Middle Ages and later, concerning the origin of senile cataract from dyscrasic and metastatic causes. I will say only that, even as late as the first half of the nineteenth century, treatment of this condition with derivative, antiarthritic and antirheumatic remedies, blisters and setons, based on these hypotheses, was held in high estimation. The recognition of the hopelessness of such attempts, together with increased clinical observation, opened new lines of thought, but yet the idea that the general condition was in some way of importance in the genesis of senile cataract could not die out, although it no longer found expression in such therapeutic measures, and although conceptions concerning the nature of this causal connection have by no means become clear. It prevails in modern ophthalmology. *Becker*, who sought to find the origin of cataract in the lens itself, gave indications that he was not forgetful of the rest of the organism. He says: "If we take the simple senile as the prototype of the primary cataract, we must see its cause in the senile sclerosis of the lens. I have tried to explain how the contraction thus produced leads to the formation of cataract. But, as not every individual, at least up to a certain age, suffers from cataract, there must be an exciting cause. Also, as both eyes of an individual are involved in cataracta senilis simplex, this exciting cause must exist in the general organism. Finally, as both eyes are never affected at exactly the same time, and, as *v. Graefe* rightly pointed out, as the second eye affords the more favorable prognosis for operation, showing that, aside from the cataract, the eye first affected is the less healthy, it follows that a cause which takes part in the formation of cataract is to be looked for in the eye itself."

v. Michel advanced another step. He believed that he had discovered an explanation of the fact that the development of cataract in the two eyes is not uniform and of the same intensity in the sclerosis of the carotid, which is sometimes bilateral, sometimes unilateral. "As both a quantitive and a qualitative change in physiological metabolism results from a retardation of the circulation in the vascular area affected by arteriosclerosis, and as there is a direct connection between the region supplied by the carotid and the circulation of the eye, it is to be supposed that disturbances in the former may exert an influence upon the nutrition of the eye, and particularly of the lens." Besides this accentuation of the importance of arteriosclerosis *v. Michel* took into account the relations of the entire organism to the genesis of senile cataract. "Cataract often forms the first link perceptible to the patient of a chain of subsequent general disturbances. Death from apoplexy is not rare. Any serious general disease, like an intercurrent pneumonia, can greatly accelerate the development of an incipient cataract." "An inadequate composition of the blood, a diminution of the nutritive material, is also of importance etiologically; such a cataract might be termed an inanition cataract, or a cataracta cachexia. There are individuals who have become marasmic through disease, excessive physical efforts, many, or difficult labors, poverty, sorrow and care." Clinical observations do not reveal a direct causal connection between senile cataract and atheroma of the carotid, but even *Becker* admits that the morbid condition of the uveal vessels is of importance in the development of cataract. It is to the credit of *v. Michel* that he accentuated this proposition. Nephritis is another factor which at one time was recognized. Although this cataract is not, strictly speaking, a simple, senile cataract, it seems best to state briefly in how far, and in what sense the relations of the general organism to senile cataract have hitherto been taken into account. *Deutschmann* was the first to claim that albuminuria could cause cataract through a change in the composition of the aqueous and vitreous, but it has been shown by numerous statistics that such a connection cannot be established. At present we can only maintain, with *Deutschmann* and others, that in this case, as in atheroma of the carotid, the degeneration of the vessel walls probably plays an important part in the development of cataract. Even *Frenkel's* attempt to clear up the connection between cataract and renal disease, by testing the penetrability of the kidneys with intramuscular injections of methyl blue, is indecisive. It may be mentioned also that according to *Hirschberg* "beneath the glowing sun of India senile cataract ripens twenty years earlier than among us."

According to *Schnitzler*, the ultraviolet rays of sunlight are of importance in the etiology of senile cataract. Yet *Hess*, in harmony with *Birch-Hirschfeld*, states impressively "that up to the present time no case of human cataract has been proved to originate from the action of light, and that conclusions concerning an etiological relation of light to the origin of senile cataract drawn from the observations of *Birch-Hirschfeld*, *Widmark*, and *Herzog*, who produced lenticular opacities in some cases with the arc light, seem at least to be very risky."

Heterochromia has been described in connection with cataract by *Hutchinson*, *Bistis*, *Schapringer* and others. The influence of heredity has been suggested often, as by *Vossius* in his textbook. The influence of a bad condition of general nutrition upon the nutrition of the lens has been spoken of very frequently, and it is sometimes stated that a cataract originated from a general disease. Such factors are held responsible particularly for the rapid advancement of a cataract. *Groenouw* has also claimed that the improper use of water cures, for the purpose of reducing bodily conditions, may promote the development of the opacity. The coincidence of prostatic hypertrophy with cataract, to which *Hogg* called attention, comes under the heading of presenile marasmus. Thus we find in literature not a few efforts to disclose relations between senile cataract and the general organism. *Groenouw* writes: "The ultimate causes of senile cataract are certainly not yet known, but one can hardly be in error if he holds responsible for its origin not a local disease alone, but a certain disturbance of the general condition as a cooperating factor." *Hess* says: "In my opinion the theory that senile cataract is connected with disturbances in the general organism has much more in its favor than the mechanical explanation from the processes of a still quite unknown pathological shrinkage of the nucleus. But, in the former theory, I think that the purely physical factor of the altered osmotic conditions should not be brought too strongly into the foreground by slighting the biological properties of the lens elements. The outer cortical fibres of the lens, at least those that show distinct signs of growth, we must consider living elements, although their vitality is low, whose changes need not end in accordance with the physical laws of dead material."

56. Senile Cataract as a Specific Disease of Metabolism

A patient with senile cataract must wait, as yet, until his vision has fallen so low that no improvement can be hoped for except from extraction of the lens, although frequent attempts have been made to check its development medicamentally. Recently it has been recommended by French ophthalmologists, *Badal* and others, to administer potassic iodide internally, and also to instill it into the conjunctival sac. German ophthalmologists, like *v. Pflugk*, also have made such attempts. We likewise try, from other points of view, to penetrate the still obscure pathogenesis of senile cataract, and to find ways and means by which to influence it. I have formulated a theory concerning the etiological relations of the general organism to it, which may be termed the biological. I will try to sketch it briefly. It is based on the teachings of immunity, because serum investigation has rendered possible an unexpected insight into many delicate processes of cellular life. When the results of the immunity research are relied upon, and the phenomena of senile cataract are followed with open eyes, the thought comes almost involuntarily that a cytotoxic process takes place in the epithelium and fibres of the lens. Then many questions arise which need to be discussed from this new point of view.

I can illustrate my ideas concerning the cause and nature of senile cataract by arranging in parallels some of the symptoms of cytolysis of the red blood cells and the processes of the development of cataract. This is only a comparison and, like all comparisons, it has its great faults. I wish to say further that the view I am about to enunciate is simply a hypothesis, the same as all the other theories to which I have referred.

It is known that we attribute to the discoplasm in the red blood cells the properties of a membrane which impedes diffusion, or, perhaps more correctly, consider the true protoplasm of the blood cell to be such a membrane, which possesses among other functions that of preventing the escape of hæmoglobin. The lens likewise has a membrane, the capsule and epithelium, which prevents the escape of its constituents. But there is this difference between them; each blood corpuscle has this device, while the capsule and epithelium perform this function for the entire mass of lens fibres.

If the capsule is opened, or if the epithelial cells are destroyed, fluid, like the aqueous, penetrates between the lens fibres, so that not merely single fibres, but large masses of them are destroyed, at least in young persons, and when the effect is sufficient the entire lens is lost. Another parallelism is this: If the hæmoglobin escapes from the blood cell and is dissolved in the surrounding fluid the physiological function of the cell is extinguished, the blood cell is dead. The disintegration of the lens fibre, the cataracta senilis subcapsularis, is nothing but a death of protoplasm, although the chain of phenomena is longer and more complicated. Just as in hæmolysis the membrane, or the discoplasm, becomes permeable, so in the development of cataract analogous, abnormal phenomena of diffusion appear between the lens and the fluid surrounding it. What occasions the death of the protoplasm? By what means is the process introduced?

A series of phenomena in the pathology and physiology of the blood cells and of the lens also may be arranged in parallels. We know that new red blood cells are constantly being formed, that others are constantly dying in the organism and that the different ages of the blood cells can be recognized from their varying resistance to foreign as well as to physiologic influences. Therefore it is positive that the blood cell reaches only a certain age and then dies. The different ages of the fibres in the lens can be perceived likewise, and we know that the oldest fibres in the nucleus can scarcely be spoken of as living cellular structures. Hence the idea is not so far fetched that cataract is nothing else than a physiological death of protoplasm, that the life of the lens cell ends sooner in persons with cataract than it does in other people. I cannot subscribe to so fatalistic a view. I am convinced that the sclerosis of the lens has nothing to do with the subcapsular cortical cataract, for in this, the commonest form of senile cataract, those parts of the lens which might be termed physiologically dead through the normal process of development, parts of the nucleus, are unaffected by the cataractous disintegration, but the seat of the disease is in the younger, cortical parts. That these do not show a tendency to die at that age is shown by the fact that the lens exhibits remarkable phenomena of growth, even in very old age. Lively phenomena of proliferation are met with in the epithelium of the lens even when the cataractous process is going on.

I have always been opposed to the idea that senile cataract is nothing but a sign that the predestined life of the lens cells expires sooner in one person than in another. We must seek for the cause of the evident death of protoplasm in the lens in senile cataract. Perhaps the lens lacks normal nutritive material. If we take a blood cell from the vessels of the body it finally dies, even in the best isotonic solution, and the lens is still more sensitive, it can survive only a few hours. But in my opinion it is doubtful if such a lack of nutriment for the lens in the senile organism is sufficiently probable to be considered seriously as the cause of cataract. The occurrence of constitutional cataract in early life, when the cause is certainly not a lack of nutritive material, makes it probable that it is not the sole cause which renders the lens cataractous in these cases. Besides, the nutritive substances in the blood and juices of the body in old age suffice for the nutrition of the lens. In addition to this, persons afflicted with cataract do not always present the appearance of marasmus, or of any other condition in which lack of nutrition can be considered seriously. It must be that, as the result of senile changes, the secretory organ of the eye does not admit sufficient nutriment into the intraocular fluids from the blood. Yet the senile changes are met with regularly in eyes in which cataract is wholly absent. There is hardly a man who does not exhibit arteriosclerotic changes in his vessel walls in old age; still there are innumerable old men whose lenses do not become opaque. If the statements are correct that in cataract an increased amount of albumin is met with in the aqueous, it is not impossible that the albumin comes not only from the lens, but also from the blood. In that case the lens may be surrounded by a surplus of the constituents of the blood in senile cataract, it may be overnourished.

Above all it must be remembered that lack of nutrition alone does not satisfactorily explain the occurrence of opacities. I have here lenses which have remained perfectly clear for months in oil, without a trace of nourishment. Therefore I find no cogent reason to suppose that the lens has to suffer and become cataractous from such a lack of nutrition. The idea becomes still more questionable when considered from the standpoint of *Leber*, that the need of nutrition by the lens is not great, that it needs additional nourishment only to maintain its growth. With a little deficit of the normal increment of nutrition in old age the most probable result would be only an abatement of the further growth of the lens. The lens may remain perfectly transparent and healthy when there is a deficiency of nutritive substances, and it is certain that these cannot be absolutely absent in old age. It needs hardly to be mentioned that a slight increase of the normal constituents of the blood in the intraocular fluids is not the cause of senile cataract. The proof of this is furnished by the fact that opacity of the lens has never been caused by the great and long continued abnormal superabundance of such substances in the anterior chamber met with after paracentesis, or in iritis.

We are thus led to the conclusion that the physiological change in the lens due to age is not the cause of cataract, that a lack of nutritive substances is not the cause, and that an increased admixture of the normal constituents of the blood in the surrounding fluids does not produce disease of the intact lens. We will first begin to understand this disease when we start from its first clinical symptom. An increased entrance of water into the lens was the first gross, if I may call it such, physical sign.

Since none of the agents which have been suggested have met with our approval, how is it that the cells can be robbed of their power to resist the entrance of water? Mechanical injury is not responsible. The blood cell may be injured mechanically, it absorbs water and lets hæmoglobin escape. The lens also may be injured mechanically, it allows water to enter and a traumatic cataract develops, but such influences are not met with in senile cataract. The same effect may be produced in the blood cell by a chemical poison much more easily than by a mechanical injury. May not the spontaneous development of a cataract be due to a direct toxic action upon the cells and fibres of the lens?

If we are willing and able to regard cataracta senilis thus, not from the points of view which have been discussed already, everything is excluded except the ascription of the origin of senile cataract to an injurious action of substances upon the lens. This is my theory: *The subcapsular senile cataract is just as truly a metabolic disease of the lens as, for example, diabetic cataract.* The only question is: What kind of substances exert a harmful influence upon the lens?

The nature of these is wholly unknown to us as yet, and their origin can only be known empirically. The possibilities are many, but it will be long before we know their chemical reactions. It seems to me just as proper to assume that metabolic products injurious to the lens are present in the organisms of men who have senile cataracts, as to assume the presence of such abnormal metabolic products in diabetes, but to prove it will probably be impossible for a long time to come. Not even in the simple poisoning of animals with naphthalin could the separate product be demonstrated which must be responsible for the origin of the naphthalin cataract. The conditions in the involution of the human organism are still more difficult. The opacities frequently met with in the vitreous show that the composition of the fluids in an eye with senile cataract is not normal, and it may be that in the future the composition of the fluids surrounding the lens may be recognized to be abnormal, even without the presence of visible opacities. At present we can only inquire what sort of harmful substances they may be.

The study of hæmolysis is again our guide. First we have to consider whether there is a large number of sharply defined chemical substances to be taken into consideration, and whether there are poisons to the protoplasm of the lens. We are acquainted with numerous chemically defined substances which are able to dissolve and destroy red blood cells. The fact is very remarkable that the action of very many poisons to protoplasm has nothing to do with the conditions of isotonia. When, for example. dilutions of 1 : 100,000 of certain saponin substances quickly change the color of the blood in a physiological salt solution to lake, it is evident that with such dilutions a change of the osmotic conditions is not the cause. I emphasize this because there may very well be poisons to the protoplasm of the lens, the action of which may be wholly independent of the conditions accentuated by *Peters* of an increase of salt in the aqueous. Such poisons to the lens actually exist, as is proved by the naphthalin poisoning of rabbits. The action of all these poisons to the protoplasm depends mainly on processes of solution, in general accordance with the laws that govern the action and distribution of most chemically defined poisons. *Ransom* discovered the fact that in the solution of the red blood-cells by saponin the cholesterin of the blood cells receives the poison first, hence the toxic action first affects the protoplasm. Perhaps the means of solution and fixation through which naphthalin and its products transmit their toxic actions to the lens, may yet be found.

Whether such chemically defined poisons to protoplasm in the human organism play a part in senile cataract is still beyond our knowledge, but we need not have recourse to their actions to explain the pathogenesis of cataracta senilis, for there are, or at least can be, plenty of other cell poisons in the human blood and blood serum. The study of immunity has revealed to us a large class of hæmolysins, which differ from the chemically defined cell poisons in that they enter into a true chemical union with certain groups of atoms of the protoplasm. Among these are the hæmolysins of the higher plants, those of bacteria, of the lower animals, of glandular secretions, and especially of the blood serum. The latter only is of interest to us at this place.

Since *Ehrlich* and *Morgenroth* discovered the fundamental fact that only such red blood corpuscles are destroyed by the hæmolysins as are able to fix these substances, these bodies of the serum have been carefully studied and this point has been perfectly proven: The blood serum may contain specific cell poisons to foreign species of cells. This fact must be correctly borne in mind, for by it we may at least try to form a conception of the pathogenesis of cataracta senilis.

We are able to increase the hæmolysins of the normal blood serum by immunization. We draw from this, for our conception, that the animal organism can contain not only specific cytotoxins formed by Nature and in physiological metabolism, but that it can also form an increased number of cytotoxins in response to proper stimulations and under certain conditions, so that its fluids are for a long time inundated with such materials. Inject an animal of species A with erythrocytes of species B; then the blood serum of species A becomes highly toxic to the blood corpuscles of species B. This phenomenon becomes still more interesting. *Ehrlich* and *Morgenroth* have shown that the organism can react with the production of specific cytotoxins to the introduction of the blood cells of the same species of animals. *Ehrlich* treated goats with injections of goat's blood and the animals formed substances which dissolved the blood corpuscles of different goats of the same species. Thus we arrive at our second important proposition: The blood serum can contain specific cell poisons not only to cells of other species of animals, but to cells of the same species.

Now comes the most remarkable fact, that these isocytotoxins dissolve the blood cells of the same species, but are harmless to those of its own body. This law of cytotoxin formation appears with certainty and absolute regularity in the destruction of foreign cell material. But as a rule, and this is in my opinion perhaps of fundamental importance for the understanding of the pathogenesis of senile cataract, this biological law of antibody formation is interrupted physiologically by the resorption of its own cell material. *Ehrlich* and *Morgenroth* have shown that ordinarily the serum of goats that have been treated with blood from other goats, and even with their own blood, is unable to dissolve its own blood cells. Isolysin, never autolysin, is formed. The normal animal organism is thus provided with regulative devices which prevent the formation of poisons deleterious to its own body cells in the resorption of its own cell material. A large part of our body cells are continually being destroyed under physiological conditions. In the resorption of this cell material the path is clear for the development of autotoxins. according to the laws that govern the formation of antibodies, unless regulative devices interfere. As these regulative devices, which limit, or entirely prevent, the formation of antibodies in the resorption of its own cell material, may take form in the individual case, the idea is not far fetched that occasionally they may and must fail.

Such conditions for the development of autocytotoxins are abundantly present in the senile metamorphosis of the human organism. If one will but consider the vast transformations that take place in an organism which is becoming, or has become senile. the changes in the skin, the melting away of the fat, the atrophy of organs. especially the involution of the genital organs, and the changes throughout the vascular system, he will acknowledge that this regressive metamorphosis may very well be reflected in the biological composition of the blood. We know from numerous facts in the study of immunity that the serum of the human fœtus differs from that of adults. We know in part the bodies which are absent in the serum of the child, though present in the blood of its own mother. I mention these facts. which may be demonstrated experimentally with ease, only because they are best fitted to guard us against the error of thinking that one human serum is just the same as another. I need only refer to the fact that the least infection, even when of no serious importance to the life of the organism, suffices to cause in certain organs of the body the production of numerous antibodies, which again differ from each other. The formation of antibodies is not effected alone by the action of bacterial products. Since we know that the introduction of cell materials, even of ordinary food, excites the development of specific antibodies, it is not a bold conclusion to assume that such processes may be excited in the organism as it grows old by the senile involution of the organs, for what the experimenter excites in the animal, when he compels the organism to resorb masses of protoplasm, is the existing condition in senile involution. The student of immunity can only wonder that in this senile process, which gives so many visible symptoms, not many grave disturbances of cell life are created, unless the symptoms of senile degeneration as a whole may seem to be a sufficient disturbance. Everything indicates that regulative devices are at the command of the organism in these processes of senile metamorphosis, on the function of which depends the length of time it is possible for a certain cell complex to continue to live. A part of these regulative processes have been recognized by *Ehrlich* in his experiments. It can hardly be supposed that the human organism, which does not differ from the animal organism as regards the formation of antibodies, should behave essentiallv in a different manner.

At the present time the substances necessary to the serum diagnosis of syphilis are considered to be in part autoantibodies to normal substances of the organism. Active immunization has proved with certainty that the human organism can form the most various new cytotoxic substances, and amass others in its serum, so it is at least possible that in senile involution antibodies originate in the blood which possess a specific affinity for an important constituent of the protoplasm of the lens. If these can no longer be kept back by the secretory organ of the eye, the lens cell may be injured by the fixation of such abnormal products of metabolism with corresponding receptors of the protoplasm of the lens, in a way analogous to that in which the blood cell suffers through the fixation of the specific cytotoxin. It is not necessary that these autocytotoxins should be substances which menace organs essential to life. Substances continually circulate in our serum which are highly toxic for certain red blood cells, but cannot affect in the least our own erythrocytes. The study of immunity, under the guidance of *Ehrlich*, has shown that a chemical fixation of the toxin to corresponding groups of cells to be poisoned is necessary in all these complicated processes. Only such cytotoxins can come into consideration for the lens as possess a specific affinity to certain groups of atoms of its protoplasm. This explains the fact that there need be no traces of the effect of a cytotoxin anywhere else in the body. That, under certain conditions, the entire organism may suffer from such changes in metabolism is shown in diabetes, in which the faulty oxidation of the sugar cannot be responsible for the cachexia.

One of the chief merits of *Ehrlich's* theory of receptors is that it explains satisfactorily the specific nature of the antibodies. We know that species of antibodies set free by cells do not exist, in the sense in which the term is used in zoology and botany, but that the specific relations of the antibody exist exclusively between the receptors and the antibodies produced by them. Thus I imagine that in senile metamorphosis the organism has to undergo a very complicated internal process of adaptation. When the man has passed the acme of his powers, and the senile involution of his organs has begun, very minute regulative devices must intervene to prevent the formation of noxious products of metabolism. If these fail to a greater or less degree, there may appear among these products of metabolism such as are able to injure the epithelium of the lens. Regressive changes in metabolism and cataracta senilis are inseparably associated, and thus the senile cataract appears to be a specific metabolic disease of the lens. This conception of cataracta senilis as a specific effect of cytotoxin is transferred very easily to the constitutional forms of cataract. Such a process seems to me to take place particularly in diabetic cataract. Diabetes is a disease in which there is a grave change in the intermediate metabolism. It is not impossible that both forms of cataract owe their origin finally to the same substances.

In support of this theory it can be shown that the secretory organ of the eye, under normal conditions, endeavors to keep cytotoxic bodies in the serum away from the intraocular fluids. Traces of them appear in the aqueous only when in immunization the blood serum is inundated with such substances, as the recent studies of *Miyaschita* have demonstrated. I have furthermore shown that only those antibodies penetrate the capsule and enter the lens for which the protoplasm of the lens cell has a maximal specific affinity. Recently *Salus* raised the objection to this hypothesis that the constituents of the lens which I call receptors are not such, but are lipoids. I think differently. The presence of receptors in the lens is evident from the discovery of *Uhlenhuth* that a specific antiserum to the albumin of the lens can be prepared, as the formation of such an antiserum unquestionably presupposes specific receptors. The experiments concerning anaphylaxis likewise furnish distinct evidence of the presence of receptors in the albumin of the lens. This is the present position of my hypothesis concerning the pathogenesis of subcapsular senile cataract. It has excited considerable discussion and I believe that it will prove fundamental.

57. Indications and Preparations for the Operative Treatment of Cataract

The operative treatment of cataract is one of the most brilliant features of ophthalmology. There is hardly another operation which is so fascinating as the extraction of the opaque lens from the eye, and every physician should have the opportunity to see a blind patient lie down upon the operating table and arise therefrom with vision restored. Such skill appertains to the ophthalmologist, but it should not be thought that the general practitioner can take no part. It is not uncommon for patients with cataract to consult their family physician first, to seek advice concerning the prospects of their condition. If they know the nature of their trouble, and they often do, they wish to know whether the cataract is ripe and ready to be operated on. The general practitioner should be able to give them adequate replies.

The indications that the cataract is ripe accepted by modern ophthalmologists are quite different from those required not very long ago. A senile cataract may be pronounced clinically ripe when the anterior chamber is of normal depth and the cortex beneath the capsule is perfectly opaque. This is the condition in which a subcapsular cortical cataract may be extracted most easily and completely. For this reason surgeons formerly waited for this stage to develop, and some do so even now. Before asepsis had entered into the practice of ophthalmology, when a large number of eyes that had been operated on were lost as the result of suppuration in the wound, a surgeon was constrained to introduce instruments into the eye as little as possible. He rightly chose to operate at a time when the cataract could be extracted best, and a subsequent intervention was least to be expected. This standpoint has been materially changed under the revolutionizing influence of antisepsis and asepsis, by which the dangers of infection have been diminished materially, so that sterile instruments may be introduced repeatedly into the eye, provided that the introduction is properly performed. Therefore it is possible for the skillful operator to remove particles of lens matter, which were not expelled with the delivery of the lens, and to perform later a second operation, when necessary. Hence it is no longer needful in any given case to await clinical ripeness of the lens, for the operation may be performed at an earlier stage.

In many cases the idea that the mature cataract is the easiest to remove completely is only an antiquated theory. Even the most skillful surgeon cannot prophesy with certainty whether a ripe cataract can be extracted in toto so smoothly as to render a subsequent operation unnecessary. Many accidents cannot be foreseen, and the development of an after cataract cannot be prevented with certainty. A secondary cataract may develop years afterward in an eye on which a faultless operation has been performed. The diagnosis "senile cataract" also includes several different forms which come under treatment in old age.

It was well known to the older generations of ophthalmologists that certain forms, such as cataracta nigra, cataracta nuclearis and cataracta punctata, often could be extracted completely when they were not ripe.

An important social and ethical reason should not be underestimated. Vision is very frequently impaired by the nature and situation of the lenticular opacities so that the patient is incapacitated for work before the cataract has become ripe, and we should not needlessly leave such an unfortunate person in the torture of uncertainty because of an antiquated clinical conception. In former years many patients passed away with their hope of a restoration of light to their eyes unfulfilled.

If the vision is impaired so that the patient is unable to work, and the prognosis is justified that vision can be adequately restored by extraction of the lens, the cataract is ready for operation.

To determine whether a good prognosis is justified, an examination must be made after the vision has been tested to learn whether the retina, which lies hidden behind the cataractous lens, functionates normally. In the normal course of events in senile cataract the retina retains perfectly its power to react, and even when it has received no distinct images for years the eye can regain normal vision immediately after removal of the lens.

The patient should be able to perceive the light of a candle in a dark room even when the lens is perfectly opaque, and should also be able to tell from what direction the light comes when its source is situated excentrically. This examination may be made with the simplest means. An ordinary kerosene lamp may be placed at one side of the patient and turned as low as possible, so that on covering and uncovering the eye he can perceive but the slightest glimmer of light. Then when light is thrown into the eye by means of the ophthalmoscope the patient should indicate promptly the direction from which the light enters the eye. In the clinic this procedure may be refined by means of the electric light and a photometer, but this test of light perception and localization may be made in the office of a country practitioner, and when he has found the condition to be satisfactory he can advise that the operation be performed, so far as intraocular conditions are concerned.

The ophthalmologist must still make some restrictions. A functional disturbance of the retina and optic nerve is not absolutely excluded even when the light perception and localization of an eye with a perfectly opaque lens appears to be normal and prompt. A senile degeneration of the macula, which can be detected only by a close ophthalmoscopic examination, is not infrequently met with in old people. This is an incurable disease of the retina that is characterized by the presence of yellow spots with or without increase of pigment. The gravity of the visual disturbance often is in striking contrast with the insignificance of the ophthalmoscopic changes. In cataract the fundus is not visible and such an affection of the retina may escape detection. Every ophthalmologist knows of cases in which after a perfect operation for cataract the vision remained essentially impaired by such changes in the macula, and both physician and patient suffered a bitter disappointment.

In the same way the ordinary clinical examination of light perception and localization may fail to detect a small, central spot of chorioretinitis, or a small retinal hemorrhage. Methods have naturally been sought for by means of which to test more accurately the operability of a cataract. *Pagenstecher* proposed to try the entoptic perception by patients of *Purkinje's* cross cobweb figure. In an intelligent patient with senile degeneration of the macula, who later developed cataract, I was unable to obtain any information concerning the foveal vision by this test, and therefore abandoned it. I hardly think it is used to any great extent.

Even a commencing atrophy of the optic nerve may escape detection by this examination. It is only when the cataract is not yet ripe, and the vision can be tested by means of stenopæic slits, that it can be learned whether the central area of the retina is probably intact.

Extraction of the cataractous lens may still be advised, even when the result of the test of light perception and localization is not perfectly satisfactory. Even if the macula is not intact, the enlargement of the visual field and the restoration of peripheral vision may be of good service. Eyes which have undergone intraocular inflammations, especially iritis and iridocyclitis, may be operated on successfully and many complicated cataracts may be removed with good results. An iritis which has run its course is no contraindication to the operation.

When the indications on the part of the interior of the eye are favorable, and the patient consents, there still remain many things to consider which relate to a careful preparation for the operation. The patient, the instruments and the surgeon himself must be prepared. We will begin with the patient.

First a bacteriological examination should be made of the conjunctiva of the eye to be operated on. Unfortunately the field of operation in the eye can never be subjected to so thorough a sterilization as is practiced in general surgery. The normal conjunctiva harbors a number of microorganisms, but it should not be left to chance whether the wound becomes infected or not, for modern bacteriological research has shown the way in which we can at least try to prevent as far as possible the danger of infection. The first step is to inform the operator concerning the condition of the field of operation. This is ignored in no eye clinic conducted on modern principles. The information must be adequate for the clinical, practical purpose. Our procedure is as follows: After the patient has been admitted and has received a bath, an assistant takes cultures from the conjunctival sac of the eye to be operated on in two tubes, one containing *Loeffler's* serum, the other serum agar. The next morning the cultures are examined. Anyone with sufficient experience can recognize the colonies macroscopically; in case of doubt slides may be prepared for microscopic examination. Beginners need to take more time. In this way the bacteriological condition of the normal conjunctiva is determined at least more accurately than when only a cover glass is prepared from the conjunctiva. When any inflammation is present its cause may be learned by the cover glass examination along the lines laid down by *Axenfeld* and others. In such cases we do not operate until the conjunctival disease has been cured. Even when the conjunctiva is apparently normal, the germs present in the field of operation should be investigated, and for this purpose the culture is the best means. Bacteriological researches, in which *Axenfeld* has borne a prominent part, have shown that in most cases the normal conjunctiva contains no virulent agents of inflammation, but that occasionally it does contain a small percentage of them. This agrees with the long recognized clinical fact that the normal conjunctiva is comparatively exempt from danger of infection. Otherwise it could not be understood how the loss after operations for cataract was only about 10% before the days of antisepsis. The eye is a part of the body in which the danger of infection is comparatively slight. Only xerosis bacilli and nonpathogenic white staphylococci are met with in the healthy conjunctiva, and if such is the bacteriological condition of a cataractous eye on the morning of operation we can usually proceed without apprehension. If suspicious germs are present the operation is delayed until the condition has been restored to normal, under treatment.

But the conjunctivæ of patients with cataract are not always normal. Diseases of the lacrymal passages are very common, especially in the laboring classes, and these form the gravest source of danger to an eye that has been operated on for cataract. Hence the lacrymal passages must be examined carefully in every case. I no longer rely on the conservative treatment of lacrymal disease before an operation for cataract, chiefly because of the results of bacteriological examinations. It can hardly be, when the discharge from the lacrymal sac is no longer purulent, but mucous, or aqueous, that the danger to the eye has been removed, as many have supposed, because virulent pneumococci can be demonstrated readily in many cases in which the secretion has not been purulent for a long time. Like many others I prefer to extirpate the lacrymal sac prior to the operation. Even then the extraction should not be performed immediately, but should be put off, sometimes for several weeks. Clinical experience, as well as the researches of *Plaut* and *Zelewski*, has shown that the danger of infection from the conjunctiva ceases some weeks after the extirpation of the sac. The time for extraction should be determined in each individual case by bacteriological examination, for the rapidity with which the conjunctivitis associated with dacryocystitis retrogresses differs in different cases. As we know that the pneumococcus is a most dangerous fiend in postoperative infections of the eye, such patients receive an injection of pneumococcus serum twenty four hours before operation, in order that every precaution may have been taken. No patient with a serious blepharitis, or other morbid condition of the conjunctiva, is subjected immediately to operation.

When conditions are normal the patient is to be prepared for operation. These preparations differ widely in different countries and in individual clinics. They are frequently excessive, not rarely insufficient. The golden mean is best.

There are surgeons who direct that before operation all the lashes shall be pulled out, that for whole days the margins of the lids and the conjunctiva shall be treated with antiseptics, that even the normal lacrymal sac shall be washed out repeatedly, that the puncta lacrimalia shall be occluded by cauterization, or by ligature, etc. That good results are obtained by such an operator, in spite of this maltreatment of the eye, speaks well, not for his skill, but for the power of resistance of the human eye. Nothing of this sort is to be seen here, or with most surgeons. If the cultures show a normal condition of the conjunctiva we operate in the morning. I cleanse the neighborhood of the eye with soap and water, then with benzin, and then cover the eye with *Fuchs*' wire shield, beneath which the patient can move his eye about freely. *Before* the operation the eyes should be kept as nearly as possible in their natural condition, in particular the closure of the lids should be performed in a normal manner. I lay weight on this point, for the closure of the lids takes an important part in the physiological self cleansing of the eye. It has been established through numerous bacteriological researches by *Bach*, *Bernheim*, *Morax*, *Dalen*, *Marthen*, and others, that the number of germs in the conjunctiva is materially increased after occlusion of an eye by a bandage for twenty four hours. The tears are not conducted away, the temperature in the conjunctival sac is raised, the mucous membrane becomes hyperæmic. All of these factors favor a multiplication of the bacteria. There is no need of producing this increase of bacteria in the field of the operation that is to be performed. It is self evident that the danger of infection in a not perfectly sterile field becomes greater if the number of bacteria present becomes larger. Therefore away with the bandage *before* the operation. Its importance after the operation is quite different from the bacteriological point of view. The patient wears the protective screen day and night. In the morning the eye is examined again and if it is free from irritation the operation is performed.

58. Extraction with Iridectomy and Extraction with Preservation of the Round Pupil

When the cultures show that the condition of the conjunctiva is normal, and there is no secretion in the eye covered by the screen, we may operate without apprehension, if we take care on our part that no danger of infection shall threaten the wound.

The technique of the preparation of the instruments varies more or less in almost every clinic. Our method is to boil blunt instruments in a $\frac{1}{2}\%$ solution of soda, dry them thoroughly with sterile towels and lay them out covered, ready for use. I lay special stress on the dryness of the instruments, particularly of those which are to be introduced into the eye.

In many clinics the instruments are placed in antiseptic solutions from which they are taken and directly used. However carefully they may be handled sufficient quantities of the antiseptic adhere to them, as any chemical test will demonstrate. Sometimes it remains on them in the form of drops. If such instruments are introduced into the eye a portion of this chemical poison must come into contact with the sensitive tissue, and I am convinced that this circumstance promotes many postoperative irritations. With dry instruments, which have no trace of such an antiseptic as carbolic acid upon them, this danger is certainly avoided. The only reason why instruments are placed in antiseptic solutions after they have been boiled is to prevent subsequent contamination with infectious germs. Such germs can come only from the air, or from our hands, or other parts of ourselves, to reinfect the instruments, but infection from the air does not occur and furnishes no reason for sterile instruments to be placed in an antiseptic solution. There remains only the danger that we will reinfect them ourselves. It is not difficult for ophthalmologists, who need not touch the wound in the eye with their hands, to avoid this danger. No one can infect dry instruments if he thoroughly sterilizes his hands and arms and then dries them on a sterile towel. Anyone who insists on having his instruments in an antiseptic solution should take them out with sterile forceps.

Cutting instruments likewise should be absolutely dry, but they are to be prepared differently. They are not boiled, but placed in absolute alcohol for two hours before and immediately after the operation. Shortly before the operation they are removed from the alcohol, rinsed off with sterile salt solution and dried with sterile towels.

I am convinced beyond a doubt that boiling for even a short time dulls the edge of our delicate knives and scissors. In making the section it can be recognized whether the cataract knife has been boiled or not, and I do not question that more complications ensue when dull instruments are used. It is an old maxim of surgery that the sharper the knife, and the less the tissues are bruised, the better are the conditions for healing. Next to dryness the most important precept in the immediate preparations for a cataract operation is faultless sharpness of the cutting instruments. All instruments are to be touched only by a nurse wearing sterile rubber gloves. Through these precautions only faultless instruments which have been properly prepared come into contact with the eye.

The preparation of our hands is rapid in comparison with what is the rule in general surgery. After putting on a sterile gown we scrub our hands with soap and water for only five minutes, then rinse them off with alcohol and then with sublimate solution. The modern general surgeon washes himself for half an hour before an operation. We have an advantage over the general surgeon in that we never touch the eye with our fingers during an intraocular operation, and so less time may suffice for us, at least when the operation is confined to the eyeball. The final error of seizing an instrument while the hands and arms are dripping is easily avoided. It is very difficult, if not impossible, to sterilize the entire arm with absolute certainty, and the fluid dripping from the arm may carry with it germs which have been loosened by the washing from an incompletely sterilized area. I have convinced myself by experiment that in this way infectious germs may spread with great rapidity to the wet arm and instruments and so be carried directly into the eye. *In such experiments it is of interest to note that the sublimate solution on the arm is not at all able to kill the germs in the short time at its disposal.* Although such an occurrence may be infrequent, this is a danger which should be avoided. It is avoided when the surgeon dries his hands on a sterile towel after the usual sterilization and operates with dry, sterile instruments, a subsequent infection of which is thus prevented. There still remains the one danger to the eye incident upon the imperfect sterility of the field of operation. Unfortunately this cannot be completely overcome. The certainty that the hands are sterile may be enhanced by wearing sterile rubber gloves, but these are unnecessary, because no germs can flow from the dry hands upon the instruments, and the eyeball is not touched by the hand. Besides, rubber gloves impede the delicate movements of the hand, at least according to my observation and experience. They cannot rightly be adopted in ophthalmic surgery.

I deviate also from the customs of general surgeons in wearing no mouth protector, or covering for the head. The wounds made in the eye are very minute, in comparison with the large ones usually dealt with in general surgery, the operations are of very short duration and during them prolonged speech is unnecessary. The experiments that have been made to illustrate the danger of infection of wounds of the eyeball from anything dropping in are open to serious criticism. The danger may be said not to exist, in comparison with that from the conjunctival sac itself.

It is a great advantage to elderly people that the operation for cataract is performed under local anæsthesia, as they are thus able to avoid the complications and sequelæ of general anæsthesia. Several drops of a sterile 4% solution of cocain are instilled at intervals into the conjunctival sac. The patient is then covered with sterile towels, leaving only his eyes exposed, after the environs of the eye have been cleansed again with soap and water. I do not touch the lids myself, that I may keep my hands perfectly free from the possibility of contamination; an assistant introduces the blepharostat, which is not touched again during the entire operation.

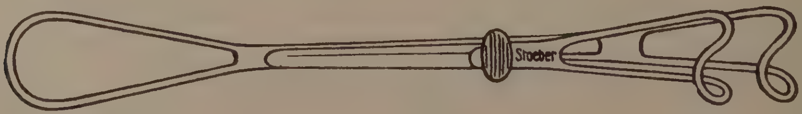

FIG. 35.—*Hess*' Blepharostat.

This blepharostat was devised by *Hess*. A small piece of gauze, cut in the proper shape, is secured between its arms so that, after the instrument has been placed in position, the lashes and lids are covered, and these breeding places of bacteria cannot be touched by the instruments, or by the hand of the operator.

The conjunctival sac is then washed out with fluid at the temperature of the body, in order to try to remove some of the bacteria and cast off epithelium. It has been disputed whether an antiseptic, or an aseptic fluid is the better suited for this purpose. It cannot be imagined that the sac, or even a small portion of the conjunctiva, can be rendered free from germs by washing it off with a 1:5,000 solution of sublimate. Such an idea is contrary to the results of culture experiments. The danger of the entrance of pathogenic germs into the eye is not reduced by this antiseptic irrigation. The proof of this is that if the conjunctiva is examined some hours after such an irrigation, the bacteria found are quite as numerous as after an irrigation with a nonantiseptic fluid, and the results obtained by those who prefer aseptic solutions are just as good as those obtained by others.

A sterile, physiological salt solution (0.85%), at the temperature of the body, is sufficient. I have no doubt that the irrigation excites a transient irritation of the mucous membrane, which, by its consequently increased supply of blood and nutritive material, favors the primary healing of the wound, but its intensity must not exceed certain limits. Such biological limits may be exceeded more readily with chemical poisons, so I prefer, for this reason also, to use salt solution. The comparatively indifferent sterile boric acid solution may be employed.

Silence is maintained so far as possible during the operation. The patient is directed to look downward continually, as the section is usually made upward at the present day.

Demonstration of Extraction with Iridectomy and of Extraction with a Round Pupil

This operation for senile cataract is known as the flap extraction. The first act is the section. After the eyeball has been seized with fixation forceps at the lower limbus and the patient has been directed to look downward, a narrow *Graefe* knife, with its edge directed upward, is introduced at the limbus and passed without delay through the anterior chamber to the place of counterpuncture, which is situated in the limbus exactly opposite the point of entrance. This straight line divides the upper third from the lower two thirds of the cornea. After the counterpuncture the section is completed upward in such a way that the incision in the cornea lies wholly in the limbus. As soon as the incision in the cornea has been made the knife is raised and a flap 3 to 4 mm broad is cut from the conjunctiva. This flap is of importance because it promotes the rapid closure of the wound and protects it from secondary infection. The second act is the iridectomy. The conjunctival flap is laid back upon the cornea, a closed pair of iris forceps is introduced and allowed to open in the eye, the pupillary margin of the iris is seized, drawn out of the wound and excised with scissors. The third act is the laceration of the anterior capsule. A closed capsule forceps is introduced, allowed to open, as large a piece as possible of the capsule is seized, torn away and removed. This clears the way for the escape of the lens. The fourth act is the delivery of the lens. A slight pressure is made backward and upward

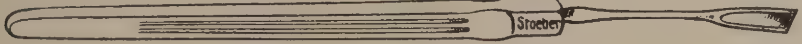

FIG. 36.—*Hess'* Lens Scoop.

upon the lower part of the cornea with *Daviel's* spoon, the wound gapes more and more, and the lens escapes from the eye. The fifth act is the toilet of the eye. Portions of the lens that have been left behind are removed from the anterior chamber with the lens scoop, the iris is replaced with a spatula so that the corners of the sphincter are situated deeply, and the conjunctival flap is stroked down into position.

If it is proposed to preserve a round pupil the iridectomy is omitted and the lens is expelled through a pupil which has been previously dilated with atropin. A so called peripheral iridectomy may be made after the extraction of the lens by seizing the iris in its ciliary portion and by making so small a peripheral coloboma that the sphincter, and consequently a round pupil, is preserved. After extraction without iridectomy a prolapse of the iris is apt to occur and to form a serious complication. The peripheral iridectomy acts as a safety valve to lessen this danger.

The patient is carried from the operating table to his bed by means of a suitable carriage.

59. After Treatment and the Normal Healing of the Wound After an Operation for Cataract

The enormous extent of the branches of medicine and the demands made on a physician at the present time do not permit much study of the history of medicine, yet we all stand on the shoulders of our predecessors and only he who knows the historical development of his specialty can perceive the aim of the mighty forward movement of our science. Details of medical history are unsuited for a clinical lecture, so it will suffice to say that up to the middle of the eighteenth century reclination of the lens into the vitreous was almost the only operation performed for cataract. Various methods were employed. That used by *Celsus* was to depress the lens into the vitreous by means of a suitable needle. *Willburg*, 1785, turned back the cataractous lens about a horizontal axis. By another method the lens was turned so that its anterior surface was directed toward the glabella frontis. The introduction of the needle through the cornea was called keratonyxis, that through the sclera scleronyxis. Itinerant couchers, who perform this operation, are still to be met with in many countries. There is something rather attractive about this quickly and easily performed operation, it restores vision at once and seems to need no after treatment.

But the lame messenger follows extremely often. Malignant cyclitis, or secondary glaucoma from the pressing forward of the iris, develops very frequently. Sometimes the lens that has been couched reappears and may even become luxated into the anterior chamber. More often the capsule of the lens is lacerated and all the complications of a traumatic cataract develop. The results were correspondingly unsatisfactory. As many as 40% of the cases were observed to result in blindness, and the uncertainty of the procedure has been demonstrated later by experiment. The modern ophthalmologist employs reclination very rarely, and there are but few indications that justify its use.

In 1752 *Jean Jacques Daviel* demonstrated that the opaque lens could be removed through a semicircular incision in the cornea, and his method of extraction prevailed for over a hundred years. In 1865 *Albrecht v. Graefe* transformed the procedure by the perfection of the modified linear extraction. The advance made by *Daviel's* extraction was enormous, but still about 10% of the eyes operated on were lost as the result of infection of the wound. *A. v. Graefe* reduced the loss to from 2 to 4%. He considered the approximately linear form of the section to be of prime importance, but we know now that the more favorable course of healing was due to the fact that the section was placed in the vascular limbus, so that the danger of infection was reduced. Since his time thousands of works have dealt with the cataract operation, which has been modified in many ways, but yet today we remain his pupils. Few of our teachers still live to tell enthusiastically of the time when surgeons came from all countries to *Albrecht v. Graefe* to be taught the new operation for cataract. May the names of *Jean Jacques Daviel* and *Albrecht v. Graefe* ever be held in remembrance.

After treatment begins the moment the operation is completed. The first act is to bandage both eyes. The eye that was not operated on is covered with our usual wet dressing, while over the other is secured a sterilized metal capsule with a central aperture, beneath which the patient can open his eye easily when he so desires. During the preparation for the operation the bandage was discarded in order that the eyes might move normally, but after the operation both eyes are covered and the patient is directed to keep his eyes closed "gently as if asleep." It is an old surgical maxim that if two fresh wound surfaces which cannot be sutured are to adhere and unite without displacement, or other complication, care must be taken that they can *rest* upon each other at first, and be disturbed as little as possible. After an operation for cataract the healing of the wound is promoted by keeping the eyes as quiet as possible, and it was shown experimentally by *Clarke* in 1899 that movement of the lids retards the closure of the wound. If our patients could promise not to move the eyes for several hours after an operation for cataract we would have the most auspicious conditions for the healing of the wound, but, when awake, the most iron will is unable to comply perfectly with such a demand.

Fortunately absolute quiet is not necessary; it is sufficient if the eyes are kept gently closed as long as possible after the operation. This is proved by the fact that a good recovery *may* take place when the eyes are left completely bare.

There are surgeons who leave the eye without any protection or dressing after an operation for cataract; they are the most extreme advocates of the so called "open wound treatment." I cannot consider this an advance in operative ophthalmology; on the contrary I am convinced that normal healing in any given case is not in consequence of the open wound treatment, but in spite of the imperfect treatment, for these operators neglect completely an important factor in medical skill—the psychical effect upon the patient.

We do not cover the eyes of our cataract patients immediately after the operation, because we believe that the bandage presses the margins of the wound together, as is sometimes thought. Such a thing is physically impossible. We cannot apportion the pressure of the dressing uniformly and only upon the two margins of the wound, and, if we should make pressure upon the eyeball, which may be compared to a sac filled with fluid, the wound would gape. Neither do we believe that the dressing can maintain an absolute, mechanical quiet of the eye. Even the quietude of the eyes when both are bandaged and no attempt is made to see is very incomplete. We bandage both eyes in order to secure an undisturbed healing of the wound by impressing upon the patient a consciousness that, in spite of the absolutely painless operation, he must consider himself ill for a few hours, if his eye is to get well. The occlusion of both eyes forms a constant monition. If the patient forgets the instructions he has received to keep the eyes gently closed, the *feeling* of the dressing is an ever present reminder to keep them quiet. Hence the primary effect of the dressing is psychical.

Bacteriological considerations have led many operators to the so called open wound treatment, but this fear of the multiplication of germs beneath the dressing is unfounded.

Whether the surgeon bandages, or does not bandage the eye the wound usually heals in the presence of and in spite of an abundant bacterial flora in the conjunctival sac. The question of infection depends not only on the presence of the morbific agent, but also, and chiefly, on its infectiosity and the condition of the organism. There is no doubt that many infectious germs come into contact with the wound and are overcome. Moreover, there is no multiplication of germs beneath the dressing during the time which is critical for the wound. The number of bacteria in the conjunctival sac does not increase for several hours, usually twenty four, and by that time, in the normal course of events, the fate of the eye has been decided, so far as primary infection of the wound is concerned. Fortunately, when the course is normal the multiplication of bacteria in the conjunctival sac comes too late to do injury.

At the first change of dressings, on the afternoon of the day of operation, a small quantity of mucous secretion may be found in the conjunctival sac and between the margins of the lids of our patient. This secretion, which has been termed cocain mucus, met with in the conjunctival sac five or six hours after the operation, contains bacteria, and yet the wound is closed, the anterior chamber restored and the eye unirritated. In order to understand why the wound heals so smoothly in spite of the presence and increase of germs in the conjunctiva, it is necessary to study the process of healing more closely.

To examine the eye twenty four hours after the operation I lift the upper lid very carefully until the region of the wound comes into view. The conjunctival flap is then seen to be slightly reddened and somewhat swollen. This redness and swelling lasts for only a few days and disappears when the union has become firm. The cornea is clear, except that in one patient there are some slight opacities on its posterior surface, which are due to the fact that the scoop, with which I removed the remains of the lens from the eye, injured somewhat the endothelium of the posterior surface of the cornea. These opacities are not serious, the rapid regeneration of the endothelium will cause them to disappear and leave no trace in from twenty four to forty eight hours.

Sometimes striæ may be seen extending from the wound into the cornea. These were formerly thought to be inflammatory, and the condition was called striped keratitis until *Hess* demonstrated that they were only minute folds in the deepest parts of the cornea and *Descemet's* membrane. After the operation the curvature of the cornea is diminished in the vertical meridian, so that the cornea is pressed together somewhat in the horizontal meridian and produces these folds. The prognosis of these striæ is good, they usually disappear in two or three weeks.

The anterior chamber is restored and is frequently rather deep. The centre of the pupil is black, with gray remains of the lens on each side. These few cases cannot illustrate the manifold pictures met with after an operation for cataract. Sometimes there is absolutely no secondary cataract, in other cases the pupil is filled with swollen masses. Small collections of blood may be seen in the anterior chamber immediately, or soon after the operation.

What strikes the observer most forcibly is the fact that the eye has adapted itself in so short a time to the changed conditions, so that its form appears to be unchanged. The loss of aqueous and the extraction of the lens suddenly diminishes the volume of the eye from $\frac{1}{27}$ to $\frac{1}{14}$. In rare cases the tension of the eye is so high that the lens is extruded as soon as the section has been made.

More often, especially in very old people, the opposite condition prevails.

The cornea may lie in folds, or form a funnel shaped depression, and even the sclera may be longitudinally folded. Then we have to deal with an unusual rigidity of the sclera, which may be noticed when the section is made. Even under such conditions the eye refills very soon and the anterior chamber is restored. This fortunate adaptation of the eye to the changes produced by the operation depends upon two facts. An increased and rapid secretion of the intraocular fluid is induced by the reduced tension and for a time the aqueous is abnormally rich in albumin, while many different antibodies pass from the blood serum into the intraocular fluid. At the same time a surprisingly rapid luting of the margins of the wound provides that the filling of the eye shall be permanent. The lens has hardly been extracted and the patient put to bed before the luting of the wound begins. This prevents a secondary entrance of infectious germs into the eye, so the wound heals perfectly undisturbed, in spite of the germs in the conjunctival sac, and gets well in the overwhelming majority of cases.

The ground substance of the cornea in the middle portion of the wound first becomes luted together by a fibrinous network. The corneal lamellæ in the neighborhood of the margins of the incision are rather swollen, while above and below the margins of the wound are separated. This swelling facilitates the primary occlusion. The epithelium of the cornea on each side begins to creep down into the wound, so that in from six to twelve hours the gap between the anterior lamellæ of the cornea has been coated over. It becomes filled in by the epithelium to the level of the cornea in from twenty four to forty eight hours.

It is not yet certain how the corneal epithelium accomplishes this. One conception is that the epithelium is displaced into the wound by a sort of amœboid movement in its neighborhood. Another view is that there is a lively new formation of epithelial cells by direct division. These two processes may be combined. At all events this rapid filling up of the wound is one of the most important factors in the prevention of postoperative infection. This epithelial cone is lifted up more and more as healing progresses, numerous cells in it degenerate and are replaced by new ones, until finally, about fourteen days after the operation, the epithelium over an unirritated wound can no longer be differentiated from the normal epithelium in the vicinity. In the meantime a lively process of healing has been taking place in the parenchyma of the cornea about the cut lamellæ, which leads to an intimate adhesion of the margins of the wound. At first many cellular elements are to be met with in the neighborhood, but at the end of three weeks the parenchyma has returned completely to its normal state. *Bowman's* and *Descemet's* membranes are not regenerated.

After treatment need not continue until the eye is thus anatomically healed. This is creditable to modern ophthalmology and a blessing to the patients.

In the sixties of the last century the dogma was taught to leave the dressing over both eyes undisturbed for three days. Today one eye is left bare at the end of twenty four hours and the benefit of changing the dressing a few hours after the operation has been demonstrated.

Above all patients are no longer kept in dark rooms, which have been responsible for many psychical and pulmonary affections. Our patients get up twenty four hours after the operation and sit quietly in a chair. Occasionally they may be led carefully about the room. After the wound has become well closed and the anterior chamber restored we need provide only that the patient does not subsequently infect his eye. The eye is washed carefully with boric acid solution morning and night, and the patient wears a *Fuchs*' shield for eight or ten days after the operation. that he may not touch his eye inadvertently or otherwise. Then for a while the eye is left bare during the day and the shield is worn only at night, because experience has shown that it is not uncommon for a patient to interfere with his eye during sleep. That the food of the cataract patient should be easily digestible hardly needs to be mentioned. When the course of healing is normal the slight injection of the conjunctiva about the wound disappears in a few days. In exceptional cases there is an increased secretion from the conjunctiva which needs to be treated with mild washes and astringents.

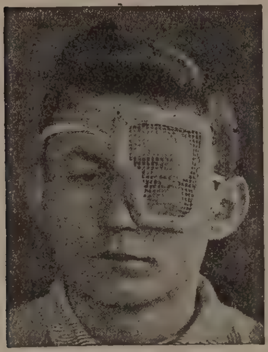

Fig. 37.—*Fuchs'* Shield.

The duration of the after treatment varies in different institutions and should be according to the individual case. We keep our patients under clinical treatment fourteen days on the average. If both eyes are to be operated on the entire treatment lasts three weeks, as I am accustomed to operate on the second eye eight days after the first. When the patient is discharged it is best to advise him to be careful for several weeks and in particular to avoid stooping and lifting, in order that the wound may not be burst open.

What has been said concerning the after treatment of eyes which have been operated on for cataract applies more or less to the after treatment of other intraocular operations.

60. Complications During and After the Operation for Cataract

Our skill has certain limits and our hopes fail to be accomplished. It is right that you should be acquainted not only with the successes, but also with the disappointments of your teacher, that you may not be too downcast by misfortune. For this reason I show you today a case in which, after a perfectly performed extraction, a purulent infection occurred in the wound and has advanced so tempestuously and steadily that the eye must be removed. Purulent infection of the wound is not the only complication with which we have to deal, and I will take this opportunity to discuss them briefly.

Complications During the Operation for Cataract

These may be divided into accidents for which the operator himself is partially responsible, and those caused by an abnormal behavior on the part of the eye, or of the patient.

If the section is too small the delivery of the lens will be difficult, if not impossible. The size of the section is determined primarily by the age of the patient, for the lens continues to grow until extreme old age. Above all sufficient space must be allowed for the nucleus. In this respect practice makes perfect.

The escape of the lens is difficult when the capsule has not been opened sufficiently. It should be borne in mind that the capsule may be thickened in hypermature cataracts.

The pressure to be made upon the eyeball must be adapted to the conditions present. The zonule may be ruptured and vitreous may be caused to prolapse by forcible attempts to deliver the lens. Vitreous is particularly liable to escape when the patient makes awkward movements and squeezes his lids together. In shrunken, hypermature, or otherwise complicated cataracts, the zonule may be defective before the operation and rupture easily, so as to allow an escape of vitreous. In all such cases further pressure upon the eyeball is contraindicated;

the lens must be removed by means of a suitable instrument, such as *Weber's* loop, or *Reisinger's* double hook.

Vitreous that has prolapsed during the operation should be cut away with scissors, and the iris should be replaced carefully; the latter procedure is difficult.

When there is a special abnormality of the eye and of the zonula the lens may be dislocated into the vitreous, but this seldom happens.

Complications After the Extraction

The most frequent, but also least serious, complication after the extraction is the simple bursting open of the wound. This is often caused soon after the operation by unquiet behavior on the part of the patient, especially by coughing, sneezing, stooping, or lifting, which may induce a sudden increase of the intraocular tension, but it may happen after the lapse of several days as the result of a blow upon the eye that has been operated on. In such cases the patient himself feels a stream of warm fluid flow out of the eye.

When nothing else has happened in the eye we find the anterior chamber empty, the tension extremely low, some hemorrhages beneath the conjunctival flap and frequently a fresh hemorrhage in the anterior chamber that has come from the region of the wound.

Fortunately such a simple reopening of the wound is not ordinarily followed by grave consequences; the anterior chamber is quickly restored and the hemorrhage in it is resorbed. But every surgeon meets with cases in which the anterior chamber will not reform for days, and the wound remains a fistula for a while.

Cases are also met with in which the closure of the wound is retarded from the first, and when the conditions are unfavorable it may not close for weeks. The reason may be that the margins of the wound are not properly coapted; frequently particles of lens fibres, threads of vitreous, or, more commonly, bits of the lens capsule lie between the margins of the wound and act like foreign bodies to prevent agglutination.

The patient needs only to be kept quiet, especially at night, with a hypnotic, like veronal. All pressure on the eye by means of a dressing is to be avoided, the protection afforded by the shield is sufficient. In cases of long continued primary delay of closure the wound should be reopened, investigated thoroughly and, under certain circumstances, closed with sutures. Some surgeons cauterize the wound, or stimulate it with tincture of iodine. It is worthy of note that this primary delay in the closure of the wound occurs more frequently after extraction without iridectomy than after the combined extraction. A dangerous sequel of delayed closure of the wound, proliferation of the corneal epithelium into the anterior chamber, will be considered later.

Incarceration of the iris in the corneal wound, without true prolapse, is of rather more serious importance. Anatomical examinations of eyes which have been operated on for cataract prove that this is much more frequent than is commonly believed. It happens in both forms of extraction through a slight, partial displacement of the iris between the edges of the wound, where it remains adherent.

In the majority of cases this partial inclusion of a part of the iris in the corneal wound is of little importance. The pupil may be somewhat displaced and the vision impaired, but on the whole the surgeon need not worry about it. Only in extensive superficial coaptations of the iris, which strangely are more common after extraction with iridectomy, the danger of secondary glaucoma is threatened by the flattening of the anterior chamber and the occlusion of *Fontana's* space.

One of the most dangerous complications met with after the operation for cataract is the protrusion of the iris between the margins of the wound so as to form a true *prolapse of the iris.* It not only interferes with the healing of the wound, but may result in the loss of the eye years afterward.

At the first change of dressing, or it may be several days later, after an apparently normal healing, a dark mass, the prolapsed iris, is to be seen between the margins of the wound. The pupil is greatly displaced and the patient often has suffered pain. Treatment depends on the extent of the prolapse, which may vary a great deal, and on the time after the operation when it occurred.

If the prolapse is small and took place soon after the extraction, we lift up the conjunctival flap carefully and try to replace the iris. This attempt is successful in certain cases. If the fresh prolapse is larger, or if the replaced iris shows the slightest tendency to prolapse again, it is to be excised with scissors, and then healing can proceed perfectly, under favorable conditions, except for a somewhat greater degree of injection in the region of the wound. If the prolapse occurs some days after the extraction and the conjunctival secretion is considerable, any attempt to replace it would place the eye in danger of secondary infection. Usually it must be excised immediately, especially when not covered by a broad flap of conjunctiva. The closure of the wound is then frequently rendered difficult, and it may be necessary to suture it, or to cover it with conjunctiva by means of a plastic operation, such as that devised by *Kuhnt.*

To illustrate this procedure I have here a patient whose globe was ruptured by a contusion and who has gone about with a large prolapse of the iris for fourteen days. The lens was extruded from the eye. After the prolapse has been radically excised the scleral conjunctiva must be used to assist in the closure of the wound. The scleral conjunctiva is dissected up over a broad area, split upward by two oblique incisions and then the flaps are drawn downward and fastened to the cornea by sutures which include both margins of the wound. In this way the gravely injured eye may be preserved with comparatively good vision.

A prolapse of the iris following an extraction of cataract always delays the healing of the wound. First it is overlaid by a fibrinous exudate and then the exposed iris is transformed gradually into a granulating tissue, which usually lasts for weeks, until the prolapse has become flattened by cicatrization and the eye becomes quiet. Even then the danger to the eye is by no means obviated permanently. Such scars do not always remain smooth, but they may undergo the so called *cystoid degeneration.* A bleblike swelling of the conjunctiva develops near the prolapsed iris which communicates with the anterior chamber and may occasionally pulsate. It is important to remember that such a cystoid cicatrix may develop without the presence of a prolapse of the iris. Such a cystoid cicatrix may readily form a serious complication. Everything may go on well for a long time, until finally the thin wall of the cyst, which is formed simply of conjunctiva, ruptures, and opens a direct way to the interior of the eye which, unfortunately, is often found by infectious germs. Thus it is that after the lapse of years a rapid suppuration of the eye may start suddenly from such a prolapse of the iris. The anatomical studies of such eyes by *Wagenmann* have shown that the suppuration of these cicatrices may infect directly the interior of the eye.

Recently detachment of the chorioid after extraction of cataract has awakened increased interest. It has been known for a long time, but *Fuchs* has demonstrated that it happens much more frequently than had been supposed.

Attention is called to such an occurrence in the eye when a few days after the operation, very rarely later, the anterior chamber appears to be shallow, or empty, without any signs that the wound has reopened. The eye is very soft. If a careful examination is made by oblique illumination a yellowish or brownish mass is to be seen in the equatorial region, which appears tense, has no folds and does not float. This sharply defined swelling may be made out still more distinctly with the ophthalmoscope. In many cases the masses are multiple. The detachment retrogresses more or less quickly, the anterior chamber again becomes deep, tension becomes normal, and vision, which may have been impaired considerably, is regained. The prognosis of detachment of the chorioid is very good; it heals perfectly and leaves no trouble. It occurs almost without exception after extraction with iridectomy, a fact which is indicative of its genesis. Anatomical researches by *Fuchs* and others (*Richter*, *Axenfeld*, *Teich*) have shown that lacerations at the root of the iris happen not infrequently during the operation. The aqueous may penetrate through such apertures in the region of the insertion of the ciliary body into the perichorioidal space, and so bring about a detachment of the chorioid. The application of a pressure bandage seems to favor the reattachment of the chorioid.

Psychic conditions of excitement, that sometimes appear in very old patients after the operation, may be called another complication. Usually they appear at night and take the form of acute hallucinations. The reasons why they appear after an operation for cataract are not yet wholly understood, but the sudden exclusion of light by the occlusion of both eyes seems to be an exciting factor. The prognosis is very good. The dressing is removed from the eye, bromide or veronal is prescribed, and the condition passes away usually in a short time. For the sake of the other patients such a case should be transferred for a few days to the clinic for nervous diseases, until a normal condition has been regained. Special attention must be paid to the protection of the eye during the psychical disturbance.

A harmless symptom, which in rare cases appears for a short time after an operation for cataract, is that after looking at a bright surface less bright objects appear to be purple, and dark objects to be of a deep green. According to *Fuchs* this *erythropsia* is due to the perception by the eye of its own visual purple.

I have seen *expulsive postoperative hemorrhage* just once. A few hours after the extraction the patient complained frantically of pain, and when the dressing was changed it was found that a violent hemorrhage had expelled the vitreous together with the chorioid and retina from the eye. Exenteration had to be performed immediately. These grave hemorrhages after cataract extraction fortunately are very rare, though they seem to happen more often in some countries. Statistics vary; according to *Sattler* they have become rather more frequent since the introduction of cocain, yet several hundred extractions may be performed before a single eye is lost in this manner. Rather more than a hundred such cases are to be found in literature, including some in which hemorrhage occurred in the two eyes from which cataract had been extracted simultaneously. The occurrence of such a postoperative hemorrhage is an indication for reclination of the lens in the second eye. The ophthalmologist cannot foresee that such a hemorrhage will occur and we are practically impotent therapeutically. Suturing of the cornea and compression bandages have been recommended, but the exenteration of the eye as soon as possible is a bitter necessity.

Glaucoma is of greater general interest. The intraocular tension must be tested with great care after an operation for cataract, but at this time transiently increased tension is not uncommonly met with. The cause of this is plausible in many cases: Swollen masses of secondary cataract, hemorrhages and displacement of the pupil may interfere with the change of fluids in the eye by contraction and partial occlusion of the angle of the anterior chamber. The same is true in those cases in which the anterior chamber is excluded from the posterior by the inflammatory formation of posterior synechiæ. Incarceration of the iris, or of the lenticular capsule, may occlude the angle of the anterior chamber and so cause increased tension. Since plausible reasons exist for the development of increased tension, we must speak of a secondary glaucoma in an eye from which a cataract has been extracted. Increase of tension may appear at very different times after the operation according to its exciting cause, a fact that warns us not to let the patients we have operated on pass too quickly out of our control. Some eyes out of a large number operated on for cataract are lost from glaucoma, as is proven by statistics.

There is one form of glaucoma after extraction that has a remarkable cause. The epithelium of the cornea proliferates through the wound and may finally line the anterior chamber, covering the posterior surface of the cornea, the anterior surface of the iris, and even involving the posterior chamber. The anterior chamber then forms a cyst lined with epithelium, and the passage for the escape of the intraocular fluid is occluded. Quite a number of eyes blinded in this way, and enucleated because of glaucoma, have been studied anatomically. The invasion of the corneal epithelium into the anterior chamber must be feared particularly when the closure of the wound is long delayed, a complication which does not always prove harmless.

Finally there are two forms of complication which cannot always be prevented by the utmost diligence and care: the *postoperative infections*, chronic iridocyclitis and acute suppuration of the wound.

Chronic iridocyclitis has been dealt with in the study of the connection between injuries of the eye and sympathetic ophthalmia. The operation for cataract is a perforating wound of the eyeball and may be followed by this complication, the same as any other injury. The important points of the clinical picture are that after an apparently normal healing the eye remains suspiciously injected, deposits form on the posterior surface of the cornea, the iris becomes discolored, posterior synechiæ are formed in mild cases, denser exudates in graver ones, opacities develop in the vitreous and the further sequelæ of iridocyclitis appear.

The picture of acute purulent infection of the wound is quite different, as may be seen in the present case. During the afternoon after the operation a more abundant secretion from the conjunctiva and a slightly smoky appearance of the cornea in the vicinity of the wound indicated the commencement of the process, and by the next morning the picture of purulent infection was present. The patient complained of not very severe pain, the upper lid was swollen, the conjunctival sac was filled with pus, the smoky cloudiness of the cornea had increased, the region of the wound had a yellowish coat, in the wound itself was a purulent infiltration, the aqueous was cloudy, and at the bottom of the anterior chamber was a purulent exudate. The first thing to do in such a case is to determine the etiology of the inflammation. In this case the bacteriological examination of the infiltrated yellowish margin of the wound revealed pure cultures of staphylococci. The majority of *postoperative* wound infections are due to pneumococci, a fact which justifies us in the hope that we may yet advance slightly in prophylaxis of this trouble by injecting subcutaneously a sufficient dose of pneumococcus serum twenty hours before operation, when these germs have been found on the conjunctiva. Unfortunately postoperative panophthalmitis of the human eye may be caused not only by pneumococci, but also by staphylococci, ozæna bacilli, streptococci, and other agents against which we have as yet no specific remedy, with the exception perhaps of the streptococcus serum. Occasional infections with such agents must be expected for a long time to come. When infection has taken place it may yet be possible to save the eye, but the result depends not alone on treatment, but also on the malignancy of the infection and the susceptibility of the patient. Benign infections may be overcome, virulent ones defy all treatment.

The most efficient means of treatment at our disposal is to reopen and cleanse the wound, to cauterize its purulent margins, to wash out the anterior chamber, to make slight incisions in the cornea and to inject salt solution beneath the conjunctiva. Introduction of iodoform into the anterior chamber has been recommended, but has been abandoned by many clinicians, including myself. All these measures are usually frustrated by the fact that we have as yet no chemically defined bodies which would destroy the microorganisms without injuring the tissues of the eye. Hence the effect produced depends in reality on their ability to induce an increase of the juices and leucocytes, which finally have to bear the brunt of the fight with the morbific agents. In this case we tried at once to master the infection, but were unsuccessful. The wound was reopened, the anterior chamber washed out with sterile salt solution, the galvanic cautery was applied to the infiltrated margins of the wound. This cauterization was repeated in the afternoon because the infection was spreading, but it could not be checked. The peripheral zone of the cornea about the wound became infiltrated and the so called ring abscess developed. The anterior chamber refilled with exudate and now, on the third day after the operation, panophthalmitis is present.

The lids are so swollen that it is difficult to open the palpebral fissure with the elevator, yet the eyeball protrudes greatly (exophthalmos) and can scarcely be moved. The conjunctiva bulbi is extremely chemotic, pus wells up from the conjunctival space, the cornea has been replaced by a yellow, opaque mass. The entire vitreous is no doubt full of pus. Fortunately postoperative panophthalmitis is much more rare than formerly, yet we must expect to lose in this manner one out of every hundred or two hundred eyes operated on for cataract. No blame attaches to a surgeon with such a percentage of loss. The patient suffers very severe pain, which will be relieved by the exenteration.

Exenteration is performed under general anæsthesia only in children and very sensitive adults; in most adults it is done under cocain. The cornea is first abscised by passing a knife horizontally through its upper third, making the incision upward, and then the rest is removed at the limbus with scissors. All the purulent contents of the globe are then removed rapidly with a large spoon, great care being taken that no traces of chorioid, etc., are left behind. The empty cavity of the sclera is then tamponed for a few days with iodoform gauze. A few days later, after the chemosis has subsided and the conjunctival secretion has ceased, the tampon is removed. The sclera then falls together, gradually shrinks and finally forms a little stump to which the muscles adhere. The prothesis which is worn later is more movable after an exenteration than after an enucleation. This is an indication for exenteration, but not the most important one. We dare not enucleate an eye in so florid a condition of suppuration, because the danger is too great that the infection will be carried through the orbit to the meninges if the numerous lymph tracts are opened. Exenteration has only one disadvantage, convalescence is usually more prolonged than after a simple enucleation.[1]

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[1] Exenteration of the eyeball is frequently performed instead of enucleation in order to obtain a better support for the prothesis, as mentioned in the text. To make the stump still better, *Mules* conceived the idea of enclosing a glass ball in the sclera. A triangular piece is removed from the sclera at each side of the abscised cornea so as to permit the aperture to be closed by a horizontal row of sutures, over which the conjunctiva is sutured vertically. Several reported cases of subsequent sympathetic ophthalmia, due perhaps to faulty technique and incomplete removal of the uvea, have proved this operation to be not perfectly devoid of danger, so that many surgeons prefer the operation devised by *Frost*, in which the muscles are separated from the eyeball, a suture is placed in each, a simple enucleation is performed, a glass ball is inserted in Tenon's space and the opposing muscles are tied together over it by means of the sutures, after which the wound in the conjunctiva is closed. This also forms an excellent stump.—F.

61. Aphakia and Cataracta Secundaria Simplex

By aphakia is meant absence of the lens in the region of the pupil. It is occasionally produced by injuries and by dislocation of the lens. Just now we are interested only in the aphakia that is produced by operations on the cataractous eye.

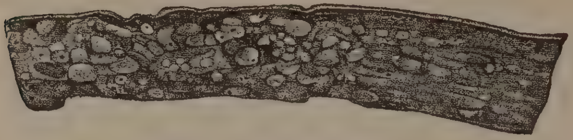


Fig. 38.—Tissue of Secondary Cataract.

A comparison of the two cases in which extraction was performed, one with a complete, the other with only a peripheral iridectomy, is instructive. From the cosmetic standpoint there is no doubt which operation is preferable. In the one case there is a comparatively broad coloboma of the iris upward, partly covered by the upper lid. In the other is an ideal, round pupil which reacts promptly to light, and the insignificant peripheral iridectomy is entirely covered by the upper lid. In such an eye the laity can see nothing to indicate that anything has happened to it, but the physician can easily detect the objective symptoms of aphakia in both cases.

1. The anterior chamber is abnormally deep and the iris is slightly sunken.
2. The iris trembles slightly during movements of the eye (iridodonesis).
3. The pupil is deep black, the physiological reflex of the lens, which makes the pupils of old people appear slightly gray, is absent.
4. The lenticular images of *Purkinje-Sanson* are absent. This is the most positive sign of aphakia.

The cosmetic reason is not the only one why the operation with the preservation of a round, normal pupil is to be preferred; the visual results obtained are better, as has been shown by statistics. Perfect vision is obtained in extraction with iridectomy in 6% of the cases, in extraction without iridectomy in over 20%. According to other statistics this difference is not so great.

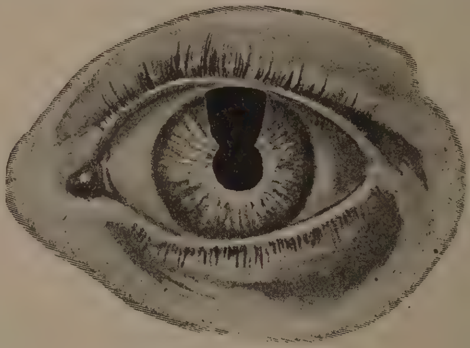


Fig. 39.—Aphakic Eye.

It is to be expected a priori that the vision will be better with a round, normal pupil, for when the pupil is small and round the retinal images are generally sharper than when it is dilated. In addition to this, the peripheral, comparatively less curved portions of the cornea come into partial use in vision after an extraction with iridectomy. But, although the preservation of a beautiful round pupil is ideal, yet extraction with iridectomy is by no means to be despised. On the contrary I believe it to be the operation which should be employed in the great majority of cases.

It likewise gives excellent visual results. Its technique is mastered more easily than that of extraction with a round pupil, though this is denied by some operators. *Becker* states, in my opinion correctly, that the greatest advantage of iridectomy is "the ease and security with which the remains of the cataract behind the iris can be completely removed by pushing, stroking and pressing manœuvres."

It is also the experience in many clinics that secondary cataract appears less often after extraction with iridectomy than after other operations. Opinions are still divided in regard to this. Technical dexterity and the kind of clinical material render comparisons difficult, and nothing is to be gained by a dispute. Our operative treatment cannot be guided by such factors alone.

Extraction with iridectomy is always preferred by cautious operators. Beyond a doubt the danger of such complications as incarceration and prolapse of the iris is less. The ophthalmologist should not always operate either with or without iridectomy; he should select his cases. Restless patients are not fitted for extraction with a round pupil. As the knowledge of the different forms of cataract advances the indications of the operation to be chosen are becoming more positive. I always operate on subcapsular cortical cataract with an iridectomy, on cataracta punctata, even when mixed with the subcapsular form, as well as on the rarer cataracta nuclearis and nigra, with preservation of a round pupil.

Immediately after the operation the patient can see somewhat about the room, but an optic correction is necessary to enable him to see objects distinctly. Prior to their disease most cataractous patients were either emmetropic or hypermetropic. With the removal of the lens the eye loses an important part of its refractive power. Such an eye presents a comparatively simple optic system. The refraction of the posterior surface of the cornea may practically be neglected and that leaves its anterior surface as the single refracting surface. In front of it is the air with a refractive index of 1, behind it is the homogeneous intraocular fluid with a refractive index of 1.3365. Under such conditions experience has shown that an aphakic eye, which formerly was emmetropic, needs a convex glass of 10 or 11 dioptres for distant vision. With such a glass vision is greatly improved even a short time after the operation, but is usually not made perfect. Fortunate exceptions are met with in which perfectly normal vision is attained after so short a time, but this is not the rule.

To ascertain why the vision does not come up to our expectations the fundus must be carefully examined first. If the retina and chorioid are intact, especially at the point of direct vision, the cause of the impairment of the vision must be sought elsewhere.

The astigmatism produced by the wound in an eye recently operated on for cataract forms a disturbing element almost invariably. Astigmatic distortion of the images can be perceived immediately with the keratoscope and the astigmatism may be measured accurately with the ophthalmometer. As the section is always made upward the corneal radius is considerably lengthened in the vertical meridian and shortened in the horizontal. It has been calculated that the mean increase of curvature in the horizontal meridian corresponds to a shortening of the radius of about 1.1 mm, and the mean diminution of curvature in the vertical meridian to a lengthening of the radius of about 0.7 mm. This difference of the corneal radii frequently causes differences of several dioptres in the refraction, which demand correction with cylindric glasses with their axes placed horizontally. If glasses are ordered for a cataract patient on the day of his discharge this astigmatism must be taken into account, but his attention should be called to the fact that the astigmatism will decrease in the course of the next four or eight weeks as the cicatrix becomes more consolidated, although it may not disappear completely, and that therefore the glasses ordered at first will not be correct later. The degree of astigmatism will be less the more peripherally situated is the vertex of the flap, so this optic factor must be borne in mind during the operation itself.

Aside from the fact that this operative astigmatism may not be sufficiently regular to permit it to be fully corrected optically, there are other causes which may impair the vision of an aphakic eye, even when the operation has been performed perfectly. The posterior capsule, parts of the anterior capsule and small bits of lens fibres are left behind in the eye. There are also asymmetries in the not round pupil, and marked signs of aberration from the peripheral portion of the cornea in the coloboma, all of which interfere with the course of the rays of light. We may therefore be very thankful when the patients we have operated on for cataract leave us with half of their normal vision; if they have more than this, so much the better.

A provisional glass may be prescribed at first to be changed at the end of six or eight weeks, but in doing this the social condition of the patient must be taken into consideration. Glasses for distant vision are not alone sufficient, the patient needs glasses for near work as well. The loss of the lens results in the loss of the power of accommodation. Most patients who are operated on for senile cataract have reached an age in which the process of sclerosis prevents the accommodative change in the form of the lens, so that the accommodation has to be reinforced, or replaced by a presbyopic glass. After the extraction of the lens a corresponding glass for near work is needed. If the emmetrope when young must accommodate 4 D in order to read at a distance of 25 cm, and when sixty years old needs a glass of that strength to read with, it is evident that, when he has been operated on for cataract and sees at distance with + 10 D, he needs for reading a convex glass of 10 D + 4 D, or + 14 D.

It has long been a subject of dispute whether the aphakic eye may not possess a true power of accommodation. It can be proved easily that such an eye can read at varying distances with a glass and it has been concluded from this fact that accommodation is present. Some have thought that the external muscles of the eyeball produced an elongation of the axis of the eye, others have contended that the ciliary muscle produced by its contraction an increase in the curvature of the cornea, and that an increase in the refractive condition of the eye was possible, especially on looking downward, but more exact investigation has revealed the sources of error that had been overlooked in the discussion of the subject.

Hess has shown that the reading of print is not a proof of an exact accommodation of the eye. We can still read with a defective accommodation produced artificially by a combination of glasses. Sometimes the pupil is covered more or less during the experiment, so that the circles of dispersion are diminished. Very often a patient who has been operated on for cataract is able to read print through a stenopæic aperture in the secondary cataract in spite of a comparatively large fault of accommodation. In addition to this, vision at different distances may depend on different parts of the bundles of rays, when such anomalies in the curvature of the cornea are present as may be occasioned by the operation, and the figures produced by dispersion are not the same in the aphakic eye as in the eye that contains a lens. These conditions furnish a natural explanation of the apparent accommodation of the aphakic eye.

Partial compensation for the loss of the power of accommodation may be secured by changing the position of the glass in front of the eye. Patients often learn to adjust their vision for different distances by moving their glasses to and fro. If the glass is moved from 1 to 2 cm from the eye the change in the refractive condition amounts to about 1 D of accommodation and makes the glass equivalent to one of +12.5 D. 3.5 D of accommodation may be obtained by moving the glass forward 3 cm.

After the optic function of the eye has been determined in this way the patient operated on fourteen days ago is allowed to go home, but not yet to pass from under the control of the surgeon.

Secondary Cataract

The result obtained by the first operation does not always prove permanent. The remains of the lens left in the eye after extraction (capsule, epithelium, bits of lens fibres) may after a while produce a new opacity which materially reduces the vision and necessitates another operation. This opacity is known as simple secondary cataract (cataracta secundaria simplex).

The clinical picture of secondary cataract is very variable. I have here a patient who was operated on for cataract a month ago. The pupil and coloboma appear unevenly gray by oblique illumination, because of a gray membrane that lies stretched behind the iris. In some places it may be seen to contain flaky thickenings. It is rather difficult to see through it with the ophthalmoscope, and vision is greatly impaired. A secondary cataract is very often much more delicate than this, and yet may be responsible for a considerable impairment of vision. In such cases the pupil may appear quite black to the cursory glance and the presence of a diaphanous membrane may be detected only with the ophthalmoscope. Such a membrane frequently exhibits a curious change of colors, and often has little folds here and there which form thickened places that prove on microscopical examination to be nests of cells.

A conception of the origin of secondary cataract may be obtained best from microscopical study. When the lens is extracted the peripheral parts of the anterior capsule, the whole of the posterior capsule and particles of the cortex are left in the eye. The particles of cortex are principally from the equatorial part of the lens because the younger fibres, which are still most firmly united, are situated at this place, and because the removal of the peripheral portion of the lens is very difficult to accomplish. Therefore a thick swelling at the equator is to be seen on the slide, which is known as *Sœmmering's* crystalline swelling. Its origin, as well as the entire formation of secondary cataract, is readily understood. After the delivery of the nucleus and the greater part of the cortex of the lens the posterior capsule comes in contact with the remains of the anterior capsule and forms a sac in which can be seen degenerated remains of fibres, epithelium of the capsule and cells which have been proliferated from the epithelium. This proliferation may be so extensive as to cover the entire posterior capsule with a pseudoepithelium.

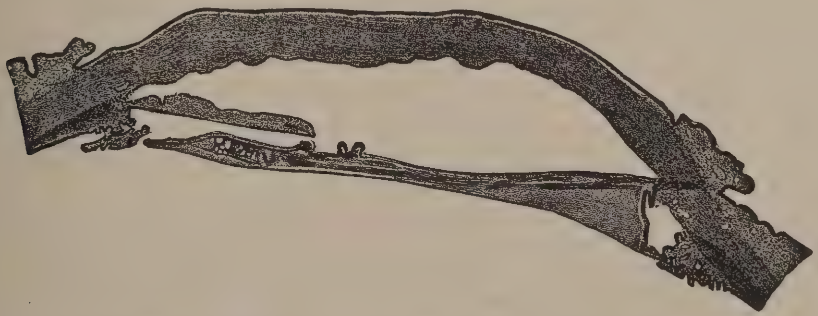


FIG. 40.—Cataracta Secundaria, from a Preparation of *Wintersteiner's*, after *Hess.*

Hence in simple secondary cataract we have to deal with a proliferation of cells from the portions of the lens that have been left behind, i.e., with an attempt on the part of Nature to institute a sort of regeneration of the lens.

The question whether the entire lens may not occasionally be regenerated more or less completely in the eye by this means, in the same way as the nail is reformed, is by no means unjustifiable. Complete new lenses may be formed after extraction in tritons and lizards, in which cells from the pars iridica assume vicariously the replacement of the lost organ, but such a restoration does not take place in man. The proliferation is confined to the epithelium of the lens itself and leads only to the formation of a more or less dense secondary cataract.

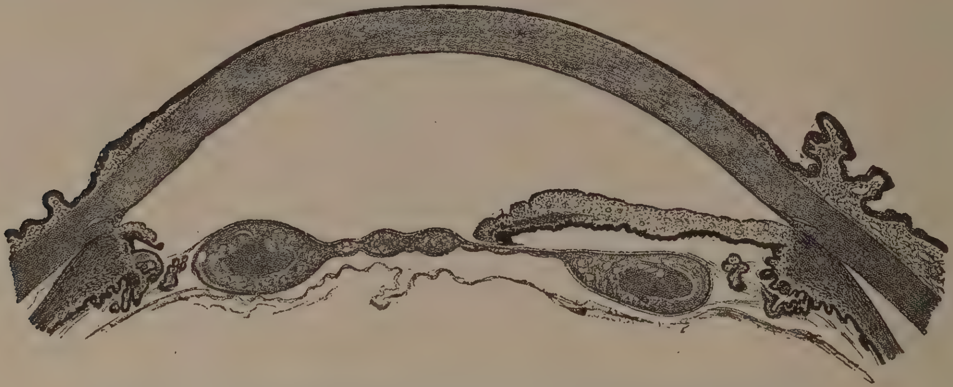


FIG. 41.—The Aphakic Eye and Cataracta Secundaria Simplex.

This secondary cataract appears in a varying percentage of cases and at very different times after the extraction. The question whether one that will materially affect the vision is to be feared after a successful extraction cannot be answered with any approach to certainty. Very often the remains of the lens are absorbed, while in other cases a more or less dense secondary cataract will develop years later and necessitate another operation.

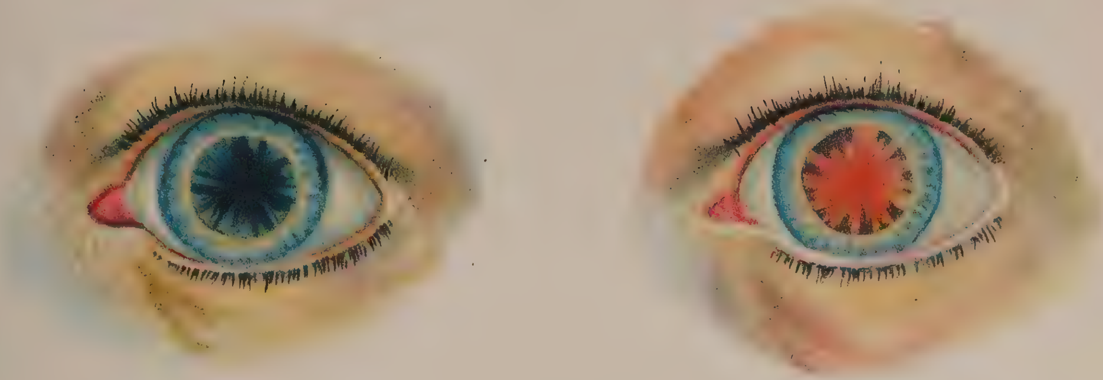
The opinions of surgeons differ widely in regard to both the indications for and the technique of the treatment of secondary cataract. This can be seen from the different percentages recorded by various operators. Some clinics have 25%, others 33% of operations for secondary cataract. Some surgeons are satisfied if they obtain $\frac{1}{3}$ of normal vision, others seek to improve even $\frac{1}{2}$ of normal vision by doing away with the most delicate membranes.

The same discrepancy prevails as to the time when a secondary cataract should be operated on. Some wait many weeks, others perform the operation very shortly after the extraction. To one surgeon the operation seems hazardous, to another to be devoid of danger.

The great multiplicity of instruments which have been recommended and devised for this operation is an evidence of the differing views and methods of treatment. I will not attempt to go into the merits of these views and methods, but will confine myself to briefly stating my own.

An eye which is to undergo an operation for secondary cataract should be perfectly quiet and should have recovered from the primary operation, so I seldom operate shortly after the extraction, except when certain economical conditions seem to demand it, especially as the resorption of the mass may progress for many weeks.

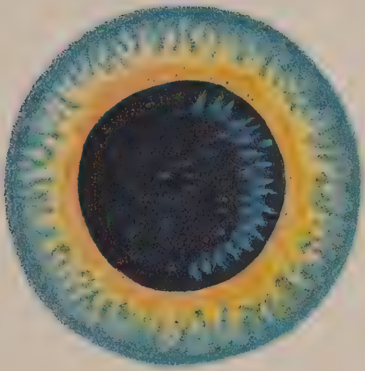
It has been recommended to incise the vitreous during the extraction in order to secure a black pupil, but I reject this suggestion on principle, together with many other surgeons, because the preservation of the vitreous is one of the most important precepts of the operation. In addition to this the number of operations for secondary cataract which become necessary later is not diminished by this procedure, while opacities in the vitreous and delayed healing of the wound may result.



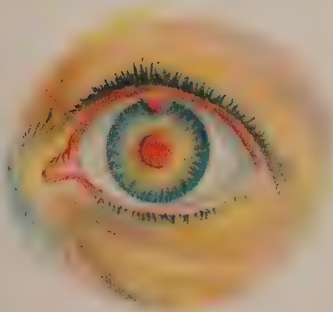
a Cataracta senilis subcapsularis

1 By direct light.

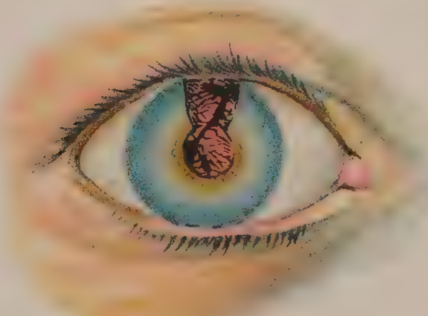
2 By oblique light.



b Cataracta punctata coerulea.



c Extraction, with peripheral iridectomy.



d Cataracta secundaria simplex.

Rebman Company, New York.

The degree of vision does not furnish a schematic indication for the operation. An old woman, who spends the evening of her life without having to work, may perhaps be perfectly satisfied with a vision of $\frac{1}{5}$ the normal, while a man who needs his eyes for his work may be obliged to have a secondary cataract obviated although he enjoys vision of $\frac{1}{2}$ the normal. *Knapp* says that an average vision of $\frac{1}{4}$ may be improved to $\frac{2}{3}$ and *Kuhnt's* experience was that average vision was present in 19.4% of his cases after extraction, in 52% after discission. The operation is not without danger and the chances must be weighed deliberately in each individual case. We will deal with its complications at another time. I usually use the knife devised by *Kuhnt* when I operate on a simple secondary cataract that is not too dense.[1]

62. Cataract after Aseptic, Contusing and Perforating Injuries of the Eye

In every injury of the eye the first duty of the physician is to ascertain whether the eyeball has been perforated or not. Every perforating wound is to be considered seriously with reference not only to the injured eye, but also to the other. If the eye has not been perforated a simple protective dressing may suffice, while in perforating wounds it may be necessary to abscise tissues from between the margins of the wound, to insert sutures, etc. But these matters have been dealt with in the treatment of injuries of the eye, and now we are interested only in the changes in the lens that result from injury. In these cases also it must first be ascertained whether a perforating wound is present, for lenticular opacities may result from both contusing and perforating injuries.

The cataract that results from contusion in man closely resembles those produced experimentally in rabbits by blows upon the eye. The opacities are slight in the anterior portion of the cortex, but more marked in the posterior, where they are frequently stellate, but may resemble nets, or leaves.

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[1] SMITH'S OPERATION FOR CATARACT

Within the past few years an operation for the extraction of cataract has been brought very prominently to the attention of ophthalmologists of all countries by Major, now Lt. Colonel, *Smith* of H. M. Indian Medical Service, and many pilgrims now visit him at his station, as they once sought *v. Graefe*, to learn the new operation. The technique of this operation, which he has performed many thousands of times, differs essentially from that of any method heretofore suggested. Its object is to remove the cataract *within its capsule*. Both a skilled operator and a skilled assistant are requisite for success. After the eye has been cocainized, the upper lid is drawn up so as to expose the transition fold, which is then thoroughly irrigated with a stream of 1:2,000 solution of sublimate. This *Smith* believes to be necessary in India, where a healthy conjunctiva is the exception, but, as he stated to the writer, he sees no reason why it should be a routine procedure in countries where healthy conjunctivæ are the rule. A speculum is introduced and a large section made in the cornea, avoiding a conjunctival flap. The speculum is then removed. The assistant then introduces a large strabismus hook beneath the upper lid and lifts it out from the eyeball, at the same time pressing up the brow with the ring and little fingers of the hand holding the hook, so as to control any contraction of the corrugator supercilii, and depressing the lower lid with the thumb of his other hand, the palm of which lies on the cheek. The patient maintains the normal position of the eye, looking upward throughout the operation. The operator holds in his right hand a smaller strabismus hook, or lens hook, in his left a small spatula. With the hook he presses on the lower third of the cornea to rupture the zonule and press the lens up through the section, while he holds the spatula ready for use, if needed, to assist in causing the lens to move by pressure on the sclera below, or to hold back the vitreous by introduction into the wound. In the majority of cases the upper zonule is ruptured first and the lens is delivered with its upper edge foremost, but in a large minority the lens "tumbles" and emerges lower edge first. The toilet of the wound consists of the replacement of the iris. The operation may be performed with or without iridectomy. It is preeminently suited for the extraction of immature cataracts.—F.

Contusion cataracts are of two forms: in the one the lens capsule is lacerated, in the other it is not. It is not always possible, or at least not always easy for the ophthalmologist to determine whether the capsule is torn anywhere or not, because the laceration may be very small and, if it lies close to the equator of the lens, it may be completely concealed by the iris. After the pupil has been dilated we should conscientiously investigate the anterior and posterior surfaces of the lens with *Zeiss*' loupe, and then again with the ophthalmoscope. Quite a number of concussion cataracts both with and without laceration of the capsule have been described by *Lawford*, *Treacher Collins*, *Fuchs* and others. Clinical experience has shown that the posterior capsule is more apt to be lacerated than the anterior in contusions of the eye. This seems reasonable because the posterior capsule is the thinner, but a great number of lacerations of the anterior capsule as the result of contusion has been collated by *Liebrecht*.

The treatment of concussion cataract may be inferred from the clinical symptoms. In most cases there is no need of haste. The eye should be kept as quiet as possible under a moist dressing, in order that the ciliary injection and the photophobia, which last for days, may pass off. If there is a laceration in the central part of the anterior capsule the swelling and opacity of the lens increases; we keep the pupil dilated with atropin and watch to see whether the rapid increase of volume of the lens excites a secondary increase of tension; if this happens the mass of lens material must be evacuated from the eye. If there is a laceration in the peripheral portion of the capsule eserin is instilled for a while, in order to enable the iris to cover and protect it as far as possible, in the hope that the wound will heal without further opacity of the lens, as it sometimes does. If the opacities are chiefly in the posterior cortex the further course of the trouble may be quietly awaited. The operative removal of the lens is always at our disposal as a last resort.

When the lens is involved in a perforating wound of the eyeball the form of cataract known distinctively as traumatic develops in the overwhelming majority of cases. The first thing then to be ascertained is whether the wound is aseptic or infected. The management of the latter class of cases has been considered already. When a traumatic cataract has been produced by an aseptic perforation of the globe the following questions have to be taken into consideration:

1. Where is the laceration of the lens capsule situated and how large is it?
2. Does the injury involve the anterior capsule, the posterior capsule, or both?
3. What is the age of the individual?

A great variety of clinical pictures are produced by various combinations of these factors, but the one most frequently met with is this:

The cornea of a child has been perforated and the wound has closed. The anterior capsule of the lens has been opened widely and gapes. The younger the child and the larger the wound in the capsule, the more rapidly does the opacity spread and involve the entire lens. The aqueous enters, and within a few hours the lens in the vicinity of the wound is opaque. The lens fibres swell, press out of the wound, often project like fungi into the anterior chamber, crumble and fall away. They may adhere to the posterior surface of the cornea and form *Fuchs*' lens deposits. This process may go on for days and weeks. New lens fibres are attacked until finally the entire lens is opaque; new flakes continually press out through the wound in the capsule. A resorption of the broken down masses of the lens takes place simultaneously with the swelling and opacification. The volume of the lens, which was greatly increased by the swelling, diminishes and under very favorable conditions the entire lens may be resorbed in this way. The pupil becomes black again, there remains only the capsule with slight traces of epithelium and fibres, and the aphakic eye can see once more. Such a complete resorption is possible only in lenses that are too young to contain firm, large nuclei. The degree and extent of the physiological sclerosis determines in great part the possibility of a spontaneous cure of a traumatic cataract and therefore the age of the patient must be taken into account. The rapidity of the development of a traumatic cataract likewise depends on the age of the lens. In very old people the lenticular opacity may be confined to the vicinity of the wound in the capsule for a long time.

A complication caused by the rapid swelling of a young lens is secondary glaucoma. If the process runs a tempestuous course and the lens swells rapidly, the physician must always take this possibility into account. The pressing forward of the iris by the swelling lens, together with the masses in the anterior chamber, may cause a temporary obstruction to the outflow of the aqueous. The tension must therefore be tested at suitable intervals, and when it is found to be abnormally high a paracentesis of the anterior chamber is indicated. A keratome is introduced and the opaque masses are allowed to escape over it from the eye. If the lens is wholly opaque a clear pupil may be obtained by this procedure. If the case is one of traumatic cataract in an elderly person the ordinary cataract section should be employed.

Spontaneous resorption is complete in by no means all cases of traumatic cataract; usually it is incomplete. After a part of the lens fibres have escaped from the capsule the wound may close more or less. This closure is favored by proliferation of the epithelium of the capsule. Then the capsule forms a sac which contains cloudy portions of the lens, epithelium and fibres, so that the traumatic becomes transformed into a secondary cataract to be dealt with in the manner already described. We are as unable to prevent it as in other forms of cataract.

The process of resorption of the opaque lens material takes place in an infinite variety of ways, but we are not always able to discover the cause of the variation. The nature of the process is not satisfactorily known. Doubtless a part of the cloudy contents of the fibres is soluble in the aqueous and is thus resorbed, but the lens contains a large quantity of insoluble material. *Wagenmann* and others have reported conditions in which remains of broken down fibres were taken up by giant cells. Moreover complicated fermentative processes in the anterior chamber and lens take part in the resorption of the insoluble constituents.

The anatomical study of traumatic cataract alone can give no information in this respect. Aseptic traumatic human cataracts present pictures quite analogous to those of cataracts produced experimentally; destruction of the elements of the lens and regenerative changes.

A total traumatic cataract is formed in the vast majority of traumatic lacerations of the lens capsule, but very lucky exceptions to this rule are met with. This fact has a practical therapeutic importance which is illustrated by the case I now present. Three months ago a splinter of iron passed through the cornea and lens of the left eye of this young man in a direction outward and upward. There was not only a laceration of the anterior capsule, but one of the posterior also, near the equator. After removal of the splinter of iron from within the eye through an incision in the sclera by means of a magnet we have waited quietly. Today it can be seen with *Zeiss'* loupe that the laceration in the anterior capsule has healed perfectly and that the anterior cortical is clear. In the posterior cortical is a slight radiating opacity which has cleared up a great deal of late, so that the eye has regained more than half of its normal vision. This case demonstrates that every injury of the capsule need not necessarily result in total opacity of the lens. Quite a number of similar cases, in which the lens has remained transparent, have been reported by *Purtscher*, *Pfalz*, *Uhthoff*, *Sacher*, *Bondi* and others. They have taught the clinician that any hasty operative intervention is contraindicated in selected cases of traumatic cataract.

The explanation of the clinical fact that the lens may remain clear after an injury to the capsule has been furnished by the experimental researches of *Leber*, *Schloesser*, *Schirmer* and especially of *P. Knapp*, concerning the closure and healing of wounds of the capsule. There can be no doubt that conditions particularly favorable to a rapid closure of the wound are present in every such fortunate case. Experimental wounds of the capsule, when not too large and properly situated, may have their swelling margins covered by fibrin, and so be protected against a further entrance of aqueous. A proliferation of the neighboring epithelium begins in a short time and the cells in the vicinity undergo a sort of displacement; these processes result in a closure of the wound. A new formation of the substance of the capsule also takes place. We have to consider the clear, newly formed lamella an excretory product of the epithelium. The lenses of many animals, especially rabbits, are much less sensitive to these injuries than human lenses, so such cases in man form the fortunate exceptions.

Much more frequently a complete opacity develops when the lens has remained clear for some time after the injury, hence we must be careful in our prognosis.

According to *Becker* grains of powder, according to *Magnus* particles of soot, according to *Leber* even copper, which can excite an aseptic suppuration in the eye, may be embedded in the lens without causing disturbance, but when particles of iron enter the lens it will undergo serious changes even though it may remain clear for a time. First a yellowish stain may appear about the splinter of iron, as described by *Graefe*. This may spread and finally give the lens a more or less brownish yellow color, rendering it opaque, while a circle of brown points appears beneath the anterior capsule. This circle has usually the diameter of a somewhat dilated pupil. *Samelsohn* was the first to describe it. Two things are to be considered in the diagnosis of this iron cataract. One is that the yellow stain of the lens may appear when the splinter of iron is not in the lens, but elsewhere in the posterior segment of the eye, even in the posterior wall of the globe; the other is that the rust brown points beneath the anterior capsule are not present in every case. Frequently this unilateral yellow stain of the lens first calls the attention of the physician to the fact that the eye contains a splinter of iron.

It is evident that a considerable portion of the iron must become dissolved, for when such a brown lens is tested with yellow potassium cyanide and hydrochloric acid it exhibits a characteristic blue color. The dissolved iron about the foreign body spreads by diffusion and is partially excreted in the form of minute granules. The chemistry of this effect of iron needs more accurate research. From the studies of *Leber*, and especially of *E. v. Hippel*, it may be presumed that the process terminates in ferric carbonate. *E. v. Hippel* supposes that there is a specific affinity of the epithelium of the capsule for the iron brought to it by diffusion in insoluble combinations. Extraction of such a cataract is to be considered only when the eye has not suffered other grave injury from siderosis of the retina. *Sattler* has pointed out that when such a cataract is extracted a peculiar condensation of the lens may be found, even when the lens is young. This form of cataract exemplifies how a lens which at first remains clear after an injury may subsequently become opaque. The same event may take place in other lacerations of the capsule which seem at first to have healed smoothly.

The opposite is also met with: A more or less extensive cloudiness of the lens may follow an injury, and then gradually pass away. Cases of this nature have been reported by *Schlœsser*, *Hess*, *Feilchenfeld*, *Bondi*, *Bresgen*, *Krueckow*, *Fuchs* and others. How this clearing up takes place is entirely beyond our knowledge. The injuries that give rise to posterior opacities of the lens which are able to pass away affect chiefly the equator of the lens. We may fancy that a number of the posterior lens fibres imbibe fluid through the solution of the equatorial cells, and then slowly regain their normal condition after restoration of the cells.

Finally, the peculiar position of injuries of the posterior capsule in relation to traumatic opacities of the lens remains to be considered. It may pass for a law that the lens is much less sensitive to injuries of the posterior, than to those of the anterior capsule. The opacities which follow ruptures of the posterior capsule develop much more slowly. *Schlœsser* proved experimentally that the vicinity of such wounds may remain clear for months. The cataract spreads only when the deeper posterior layers of the lens are involved in the injury, and even then its development is comparatively very slow. The lens fibres may partially swell out into the vitreous without important change. It is remarkable that wounds in the posterior capsule are also healed by the activity of the lenticular epithelium at the equator. The epithelium proliferates along the posterior capsule and covers it with a layer of pseudoepithelium. Such lacerations of the posterior capsule and such proliferations of the epithelium have been found by *Hess* in the cataracts of guinea pigs.

63. Cataracta Diabetica and Other Constitutional Forms of Cataract in Tetany, Ergotin Poisoning and Among Glassblowers

The first of our two patients is a marasmic young man who has to be led because he is blinded to such a degree that he is unable to find his way about alone. He tells us that a few weeks ago he was able to see very well with both eyes, and that then he began to lose his vision rapidly, not suddenly. The primary interest excited by this patient is in his general condition, but we will consider that at present only with a view to determining the etiology of the changes in the eye.

Both eyes are free from injection or irritation. The anterior chamber is rather more shallow in one eye than in the other; in each the lens is perfectly opaque. By oblique illumination the opacity appears uniform and bluish white, with distinctly radiating sectors that have a mother of pearl lustre and extend inward from the equator, with their apices directed toward the centre. No transparent cortex can be detected beneath the capsule.

Perception of light and projection are perfect, yet there is something peculiar about this cataract. The patient formerly had good eyes, so the cataract is not congenital, and yet while still young he has developed what resembles a subcapsular cortical cataract spontaneously and simultaneously in each eye. If the etiology of this cataract cannot be seen it may perhaps be smelt, for it does not require a very acute sense of smell to detect a strong odor of acetone about the patient, which arouses the suspicion, which is correct, that this is a grave case of diabetes. The urine must be examined immediately in such cases; the urine of this patient contains more than 8% of sugar, and this confirms the diagnosis of cataracta diabetica.

The other patient is of an age when senile cataract is apt to occur. The examination of the urine reveals the presence of a small quantity of sugar. After the pupils have been dilated the signs of incipient senile cataract may be seen in both eyes; gray radii extending from the equator into the posterior and anterior layers of the cortex. With *Zeiss'* loupe it can be seen that they have the same dirty gray brown color that characterizes the opacities of senile cataract. It can neither be affirmed nor denied that this cataract also is due to diabetes.

Diabetic cataract is a disease of the outer layers of the cortex of the human lens, exactly the same as the most common form of senile cataract. Its clinical appearance depends chiefly on the age of the patient and the rapidity of its progress. It develops very rapidly in a young lens; it starts at the equator and involves very quickly the soft cortex, so that the diabetic cataract presents a great resemblance to a rapidly swelling traumatic cataract through its mother of pearl lustre.

In older persons the process is slower, and diabetic cataract appears under the same form as senile cataract, from which it cannot be differentiated in elderly people.

It must not be thought that diabetes always causes disease of the lens. The statements as to the frequency with which cataract is met with in diabetes vary enormously, from 1 to 20%, because it has been discretionary hitherto to consider a cataract met with in an elderly patient suffering from diabetes as diabetic or not. The diagnosis is certain only when the diabetes and the cataract are associated in early life.

The question of the dependence of cataract upon diabetes is complicated by the fact that other forms of cataract are met with in diabetic patients. I have repeatedly found cataracta punctata in them, a form which usually lasts for decades and has nothing to do with the general disease which developed later, but anyone who is not accustomed to study and diagnosticate this form of cataract may naturally suppose that a connection exists, and deceive himself concerning the frequency of diabetic cataract.

A subcapsular disease of the cortex may be associated with a cataracta punctata that has existed for a long time and, if the patient has diabetes, it cannot be determined whether this is due to the diabetes, or is a senile symptom. The diagnosis of diabetic cataract is very difficult in old age, and we can therefore speak of but one cataract as due to diabetes.

Other clinical signs have been sought to indicate diabetic cataract in elderly persons. Diabetes may be suspected when myopia develops suddenly during the fifties. Sometimes the lens remains clear, in other cases a lenticular opacity follows this change of refraction sooner or later. Various theories have been advanced to account for this increase of the refraction. *Appenzeller* believed that the index of the aqueous was increased, but this was disproved by *Hess.* The aqueous must have a concentration equal to that of a 20% solution of sugar to attain the index of refraction of the cornea, but in such a solution the lens would become opaque and shrunken immediately.

There is no doubt that we have to deal with a so called lenticular myopia in the myopia due to diabetes; the question is only through what changes in the lens the increase of refraction is produced. Perhaps the lens may absorb an increased amount of water and so increase its curvature, but no satisfactory measurements of the curvature of the lens at the anterior pole are extant. Some, like myself, assume that the refracting index of the lens cortex is lowered by the entrance of fluid, and that the effect of this is to increase the total index of the lens.

It does not seem to me a matter of doubt that an increased quantity of water can be present in the cortical layers of the lens for a long time without necessarily producing cataract. *Salffner's* researches concerning the effect of intravenous injections of salt show that the lens can be deprived of water without becoming opaque, and therefore it may remain transparent when it contains varying quantities of water. I, myself, was able to determine in a case of rapid senile development of lenticular myopia without cataract that the anterior reflex from the cortex was weak, while the reflex from the nucleus was very strong. According to *Hess* this phenomenon indicates a reduction of the index of refraction of the cortex, but this rapid increase of refraction is not characteristic of diabetes. Anyone who lives in a country where nuclear cataract is frequently met with can easily convince himself that a rapid development of lenticular myopia is not associated with diabetes in most cases, and that it may be an intermittent stage of cataracta nuclearis. Cases have also been reported in which a decrease of the refraction of the eye was ascribed to diabetes, and for these such different causes have been assigned as increase in the refraction of the vitreous (*Landolt*), softening and atrophy of the eye (*Horner*), and latent hypermetropia becoming manifest (*Schmidt-Rimpler*). The last explanation does not apply to old people in whom the manifest hypermetropia is the total, so again it is evident that a positive diagnosis of diabetic cataract is possible only in early life, and that in later years a probable diagnosis is all that can be made.

In youth the course of this form of cataract is always typical. It is bilateral and may develop with extreme rapidity. Cases have been met with in which the lenses became opaque in a few days, or even hours. In no other form of cataract is there so little doubt that it arises from a constitutional cause, from changes in the general organism. Its connection with diabetes has been acknowledged since the thirties of the last century.

Very divergent views have been expressed concerning the origin of this form of cataract. The idea of *Claude Bernard* that the concentration of the blood is increased in diabetes was disproved long ago. The theory that the opacity is due to marasmus cannot be accepted, because the cataract may develop when the nutritive condition of the patient is good. *Heubel* believed that the sugar in the aqueous and vitreous caused the primary injury of the lens by the deprivation of water.

The processes which take place when fresh animal lenses are placed in strong solutions of sugar are entirely different from those that happen in diabetic cataract, because opacities appear in these lenses only when strong concentrations of sugar are employed. In the gravest forms of diabetes, in which the urine contains 8% or more of sugar, only very small quantities of sugar, from 0.2 to 0.4%, are found in the human eye. If the experiment is made with intraocular fluid which contains such a small amount of sugar the lenses do not generally become opaque.

Furthermore, human diabetic cataract commences as isolated diseases of individual cells and fibres. This point has usually been overlooked. In addition to this it is not a decrease, but an increase of water which is present in diabetic cataract. The second essential difference between the opacities of the lens produced in concentrated solutions of sugar and in diabetes is that the former can retrogress completely, because they are occasioned by osmotic differences of concentration alone, while in diabetes the cells and fibres of the lens are destroyed by a complicated process of degeneration, to which is to be added signs of proliferation, which are unable, however, to recall the dead cells to life.

Deutschmann advanced the correct view, that in diabetic cataract there is a local death of cells through which an abnormal process of diffusion is introduced.

It is not the sugar itself that injures the lens; the quantities of sugar met with in such lenses are very slight, and sugar has been found in perfectly healthy lenses in experimental pancreatic diabetes.

It is necessary to assume that specific products of metabolism are formed in the organism, which injure the cells and fibres of the lens in a manner analogous to that which we imagine takes place in senile cataract. I go farther than most ophthalmologists and believe that senile and diabetic cataracts are produced by one and the same cytotoxic product of metabolism. In this way only can it be comprehended how it is that diabetic cataract differs in no respect anatomically from the senile subcapsular cortical cataract. Thus only can it be understood why we cannot differentiate a purely senile from a diabetic cataract in an elderly patient. It is not the sugar of diabetes as such that produces diabetic cataract, for if it were, cataract would be much more frequent in diabetes than it is, even though we admit that the vascular changes necessary for the access of the abnormal products of metabolism to the lens need not take place uniformly. I assume that the specific substances which lead to injury of the lens may be formed in connection with other abnormal products of the intermediate metabolism in only a small percentage of cases of diabetes. These substances may also be formed in other metabolic changes, as, for example, in senile involution of the organism. According to this view diabetes is not a direct, but an indirect cause of the development of the cataracts that are met with occasionally in its course. It remains for the future to bring forward a more accurate explanation of the etiology of diabetic cataract.

The same changes are to be seen under the microscope as in subcapsular cortical cataract. Areas of degeneration are met with in the epithelium of the capsule, the nuclei of the cells are in part wanting, in other places are not uniformly stained. A similar unequal staining may be seen in the region of the equator, where fibres also appear that have undergone molecular disintegration, separated by masses of coagulated albumin. Signs of proliferation are also to be met with in the epithelium. In short, the processes are quite analogous to those in subcapsular cortical cataract, and diabetic cataract is evidently a disease of the lenticular cortex.

Changes in the pigment layer of the iris are met with in diabetes with great regularity. *Snellen* and *Hirschberg* long ago called attention to the fact that the aqueous becomes stained black very frequently when an iridectomy is performed on the eye of a diabetic, because masses of pigment seize the opportunity to float out from the iris. The reason for this was discovered by *Becker*, *Sattler*, *Deutschmann* and others to be due to changes which may be recognized under the microscope.

The cells of the retinal pigment layer of the iris are enormously swollen and have become transparent, though ordinarily they are black and opaque. The pigment is scattered over a large space. It can be readily understood that in such a condition of hydropic degeneration the pigment can easily escape.

It is very doubtful if this condition has any directly etiological connection with cataracta diabetica, for it is met with as well in eyes that have perfectly transparent lenses.

In such a constitutional disease as this, which usually reacts promptly to regulation of the diet, the question naturally arises whether antidiabetic treatment would not act favorably upon the incipient opacities of the lens. Most ophthalmologists are as yet very skeptical in regard to this, although diabetic opacities have been observed to clear up by *Seegen*, *Gehrhard*, *Nettleship*, *Gayet* and *Koenig*. Attention has been drawn particularly to the clearing up of diabetic cataracts after the use of Carlsbad water. The decision of this question will be difficult, even in the future, for it must be taken into consideration whether the improvement, especially of the vision, is to be referred to a change in the lens, or to a betterment of a coincident disease of the retina. If the patients are elderly we do not know whether the cataract is senile or diabetic, in case these are admitted to have separate causes. I have myself observed, though only in a single case, a positive retrogression and disappearance of the water radii in a woman with diabetes. Such cases are very rare.

Extraction of the lens is the only recourse in cases of advanced and mature diabetic cataracts. We have to consider the prognosis of the operation entirely aside from the question whether the cataract in question is diabetic or not. Formerly, especially before the days of antisepsis, the prognosis in diabetics was so bad that such a cataract was considered a noli me tangere. In general surgery also major operations upon diabetics are undertaken with great reluctance, because of the dangers of general anæsthesia, of coma diabeticum, and of the tendency of diabetics to infection.

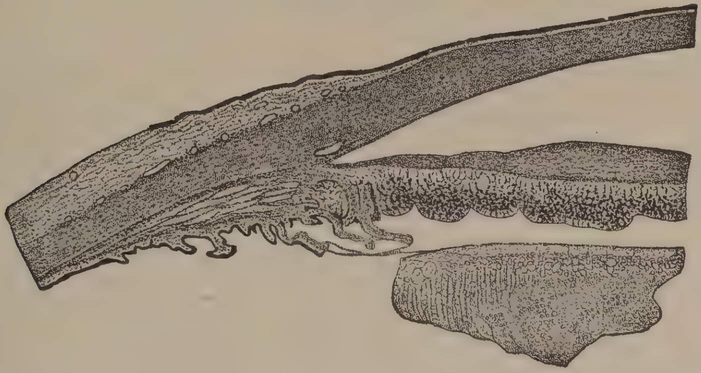


FIG. 42.—Changes in the Pigment Epithelium Layer of the Iris in Diabetes.

The conditions are somewhat different in ophthalmology. Since the introduction of antisepsis numerous observations have taught us that the little wound made in the operation for cataract may heal just as well in the diabetic as in the normal eye, and most ophthalmologists do not hesitate to operate on these cataracts by the same methods that are employed for other forms of cataract in persons of the same age. Yet certain complications must be taken into account.

Iritis, even with hypopyon, occurs more frequently in diabetics than in other patients. This agrees with the fact, that has been established both clinically and experimentally, that the tissues are decidedly more sensitive to infections in diabetes. The infectious agents themselves readily become virulent in the diabetic organism, because the power of resistance they have to overcome is imperfect.

According to my researches this iritis is always of a bacterial nature. It has been thought that the purulent iritis, which occasionally develops spontaneously in diabetes, may be caused by pathological metabolic products, but this has not been proved by careful and sufficient bacteriological study, and I believe it to be metastatic. It is self evident that peripheral neuritis and toxic amblyopia, which occur in diabetes, cannot be classed etiologically with purulent iritis and cannot be held to indicate that the iritis is of toxic nature.

Hemorrhage into the anterior chamber is another complication met with after extractions in diabetes. Hemorrhages occur into the retina and vitreous rather more frequently, in from 2 to 5% of the cases. Arteriosclerosis is often met with, and occasionally glaucoma is associated with the hemorrhages. Still none of these complications are very frequent and the results of the operation for cataract in diabetes on the whole are very good. In a large number of cases *Uhthoff* obtained good vision, 1 to $\frac{1}{3}$, in 68%, useful vision, $\frac{1}{4}$ to $\frac{1}{10}$, in 18%, poor vision, below $\frac{1}{10}$, in 14%.

Most ophthalmologists do not think it necessary, or even desirable, that the diet should be so regulated before an operation for cataract that the excretion of sugar is reduced. Even the presence of acetone and acetoacetic acid is not a contraindication to the operation. A sudden reduction of the sugar at the time of operation is not desirable. We therefore operate on our patient in spite of the grave diabetes, and its bad prognosis quoad vitam, in order that he may at least enjoy the sunlight during the short time he has to live.

Other Constitutional Forms of Cataract

Cataract after Tetany, in Ergotin Poisoning and in Glassblowers

It should be mentioned that lenticular opacities have been observed to develop in other constitutional diseases. They have been met with after meningitis in young persons, after typhus, variola, scarlet fever, chlorosis and great losses of blood, and have received the name cataracta cachexia. Their cause is still unknown.

During the past few years a form of cataract etiologically connected with tetany has attracted considerable attention. Some ophthalmologists, such as *Logetschnikow*, *Rosenblatt*, *Sæmisch*, *da Fonseca*, *Landsberg*, *Schiller* and *Wagenmann*, have noticed for a long time that lenticular opacities are not rare in people who have suffered from convulsions, and, as the clinical picture of tetany is increasingly studied, it becomes more and more evident that it is responsible for a certain form of cataract. It is met with frequently in comparatively young women who exhibit other trophic disturbances of purely epithelial formations, such as the falling out of the hair and nails, in addition to convulsions. These lenses have strikingly large nuclei. Their ultimate etiology is still unknown. *Peters* surmised that the access of the normal nutritive material to the lens might be prevented in tetany by spasm of the ciliary muscle, and that this might result in cataract, and it is probable that complicated nutritive disturbances of the lens, in consequence of a morbid composition of the blood, are present in the cataract of tetany. This is indicated also by the results of experiments on rats.

A large number of cases have been met with in Russia during the last decade in which cataract followed poisoning with ergotin. The time at which the cataract appears after the poisoning varies greatly, sometimes months or even years intervene. It is observed chiefly in the convulsive form of ergotin poisoning, and has therefore been thought to be connected with the spasm. Cataract has not yet been produced experimentally with ergotin.

It has been known for over a century that prolonged exposure to fire may injure the lens, and that glass makers are quite subject to a form of cataract known as glassblowers' cataract. This form develops very slowly and we have as yet only hypotheses to account for its genesis. As glassblowers often work in high temperatures (50 to 55°), it has been suggested that the deprivation of the body of water by transpiration might lead to an increase of the molecular concentration of the aqueous and to opacities of the lens through the great evaporation of water from the surface of the cornea, especially when continually repeated, but lenticular opacities produced by evaporation of the aqueous, and by salt, are always confined to the pupillary region, recover quickly and follow a course entirely different from that of glassblowers' cataract, so this cannot be the only cause. The fact that glassblowers are especially apt to develop this form of cataract, while workmen in other branches of industry, in which the body is exposed to prolonged heat, are exempt, indicates a possible intoxication of the body in glassblowers. The chemical light rays may be responsible. *Cramer* states, among the clinical peculiarities of this form of cataract, that in artificial ripening the anterior capsule may rupture easily.

A connection between goitre and cataract was suggested by *Vossius*, but more complete statistics fail to establish it.

It is evident from our knowledge of naphthalinic cataract, in which an intoxication of the body leads to injury of the lens, and of the forms that have been described, that autointoxication of the body may produce cataract. The nature of the process is a problem to be solved in the future.

64. Experimental Forms of Cataract Consequent Upon Physical Changes of Condition, Mechanical Degenerations of the Epithelium of the Capsule and Poisoning of the Lens

It is desirable and necessary in many ways to supplement clinical instruction by experiments; therefore this hour has been reserved for some experiments which have served to enlarge our knowledge concerning the morbid changes that take place in the lens. Lenticular opacities may be produced experimentally in three ways:

1. By changes in the physical condition.
2. By mechanical degeneration of the capsular epithelium and of the fibres.
3. By poisons.

A. Lenticular Opacities Caused by Changes in the Physical Condition

Under this heading are included the changes produced by cold, heat, and salt.

I. Opacification of the Lens by Cold

The transparency of the healthy lens is lost at certain temperatures. If lenses from young calves and pigs are placed in physiological salt solution in the refrigerator a milk white opacity appears in them, especially in the region of the nucleus. If they are then replaced in a warm salt solution the opacity disappears completely, showing that it is due to a change of physical condition, not to a degenerative process. The opacity commences at a temperature of 10 or 12°.

The picture is very similar to that of nuclear cataract, but the condition is not the same. When the fluid is warmed the opacity passes away completely in a few minutes and this experiment may be repeated as often as is desired. *v. Michel* demonstrated the dependence of this opacity upon the temperature by producing it in the living eyes of kittens by the application of ice. The opacity clears up from the periphery toward the centre, but the innermost cloudiness of the nucleus divides into three round spots before it disappears; the clear bands between them show that the components of the posterior stellate figure of the calf's lens are arranged so as to meet in three angles of 120°.

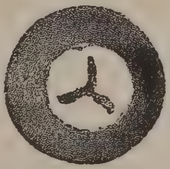


FIG. 43.—Opacification of the Lens by Cold.

The interesting part of this experiment is that by it physicochemical differences between the nucleus and cortex are made apparent, which could not be demonstrated otherwise in such young lenses, for it is always the nucleus alone, and only in young lenses, which is clouded by cold, while the cortical layers remain clear. Of course the entire lens may be made opaque by freezing. It is then as white as snow and can recover entirely when warmed.

II. Opacification of the Lens by Heat

At certain temperatures, above 45°, the albumin of the lens begins to coagulate and at higher temperatures the lens is transformed into a white mass. This opacification by heat, or coagulation, is permanent.

III. Opacification of the Lens by Salt

If fresh animal lenses are placed in a 2 or 4% salt solution they commence to become opaque at once. The anterior can be distinguished immediately from the posterior surface of the lens because the opacity is more distinct where the surface is covered by epithelium, and the boundary of the capsular epithelium is defined quite sharply at the equator. Numerous round, clear drops may be seen beneath the anterior capsule and the stellate figure of the lens begins to become visible. On the posterior surface there are no drops and the opacity is more uniform. It is remarkable that this salt opacity ceases on the anterior capsule when the lens is carefully massaged, by means of which all sorts of figures may be caused to appear.

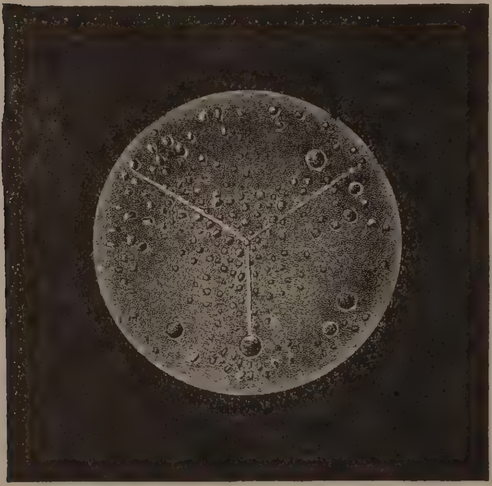


FIG. 44.—Opacification of the Lens by Salt, after *Hess*. Drops Clear as Water Beneath the Capsule.

If the lens is left for a longer time in the strong salt solution the capsule begins to become raised by the water that has entered the lens and an opacity develops in the nucleus, which may be caused to disappear by warming the lens.

These opacities may be produced not only by common salt, but also by numerous other salts and by concentrated solutions of sugar.

All of these opacities are caused by osmotic deprivation of water, not by a special chemical action of the dissolved salt, for if the strength of the solutions of these substances in which the lenses remain clear and do not change in weight are determined, as by *Manca* and *Ovio*, such solutions are found to be isosmotic, i.e., to have the same molecular concentration. Opacities produced in the lens by salt are therefore primarily direct results of the molecular concentration of the solutions employed, and are produced by the withdrawal of water. The optic phenomenon of the opacity itself has not yet been fully explained, for if the water is withdrawn quite slowly and in a proper manner the lens will remain perfectly transparent, and when large quantities of salt have been introduced into the veins of a rabbit for some time the lens may lose 7% of its water without becoming cloudy. This has been demonstrated by *Salffner*.

Ewald observed the formation of lenticular opacities confined to the pupillary region in dead rabbits, the eyes of which had been exposed to evaporation. The origin of these must be ascribed to an increased concentration of the aqueous.

Opacities of the lens may also be produced in the living animal by salt. If large quantities of salt are introduced into the lymphatic sac of a frog, or if a dense solution of salt is applied to the conjunctiva of a rabbit, its lens becomes cloudy. The chief value of these experiments with salt on the living animal is to teach us that such a molecular concentration as is necessary to make the lens opaque does not exist in the organism under natural conditions. Slight fluctuations must take place in the amount of salt contained in the intraocular fluid, but the lens adapts itself to them.

The lens is much more sensitive to a diminution of the molecular concentration, so far as the restitutio ad integrum and the preservation of the vitality of its cell elements are concerned. In this respect it acts just like a blood cell. As blood cells in hypisotonic solutions readily give up hæmoglobin, so the lens in such a solution gives up albumin, and then neither can resist the entrance of water any longer, but die very quickly.

If a strong solution of salt is injected into the vitreous of a rabbit a salt cataract develops at the posterior pole of the lens and then clears up completely, in a manner quite analogous to that in which an opacity of the lens produced by salt in vitro passes away, leaving no serious anatomical changes.

B. Opacities of the Lens Due to Mechanical Degeneration of the Capsular Epithelium and of the Fibres

Opacities may be produced in the lens by mechanical injuries of its cell elements.

I. Massage Cataract

In 1883 *Foerster* suggested to open the anterior chamber and to massage the lens through the cornea in cases of unripe cataract in order to make the cortex fully opaque. This method of maturation was much employed for a time.

If the anterior chamber of a rabbit is opened, the aqueous allowed to escape, and then the lens is massaged for several minutes by rubbing it with *Daviel's* spoon the lens will show on the next day a slight opacity in the anterior cortex and a more pronounced one in the posterior.

In the early stage of a massage cataract radii as clear as glass can be seen to extend from the equator. The fact was established by *Salffner* that such a massaged lens may increase more than 20% in volume and weight in the course of twenty four hours.

Anatomical researches, first instituted by *Hess*, have proved that the connection between the lens fibres is broken up by the massage and crushing of the lens, even when the capsule remains intact, and that much of the epithelium of the capsule is destroyed. Numerous irregularly degenerated cells may be seen on such a capsule together with signs of proliferation and mitoses. The effect of the mechanical injury is that the epithelium can no longer perform its duty, water enters the lens, and abnormal processes of diffusion are instituted between the lens fibres and the surrounding intraocular fluid. In a rabbit a massage cataract is capable of great involution and the capsular epithelium often proliferates over the posterior capsule.

II. Contusion Cataract

Cataract may also be produced by blows upon the eye. In order to understand this form of traumatic cataract in man it is essential that we should be acquainted with the part played by the epithelium for the preservation of the lens in this condition. To misapprehend the protection afforded by the epithelium is to fail to understand many diseases of the lens.

A closely related experimental cataract, known as contusion cataract, may enable us to understand better that which appears in the human eye after contusion. If the eye of an animal is beaten for several minutes, according to the method adopted by *Voelker*, *Raech*, *Hess* and others, lenticular opacities may be observed after a short time. In rabbits these usually appear in the vicinity of the posterior horizontal suture, but may also begin at the equator. Radii as clear as glass may also be seen. Opacities of this kind clear up very readily when they are of mild degree. *Hess* found at an early period numerous caryocinetic figures in the epithelium.

The vitality of the epithelium of the lens may be injured and destroyed in still other ways, especially by interfering with the circulation of the blood in the eye. *Koster* produced cataract by ligation of the vertebral vein, and *Wagenmann* saw equatorial opacities appear in the lens some days after section of the long and a portion of the short ciliary arteries. *Herrnheiser* observed a similar condition after embolism of the arteries of the ciliary body. It can be understood readily that the lens cells must die when deprived of their normal nutrition to a considerable degree.

III. Cataract from Lightning

It is not an infrequent occurrence that men who have been struck by lightning develop, among other disturbances of the eye, an opacity of the lens. The clinical picture presented in such cases is very diversified as to the situation, extent and development of the opacity. Both the anterior and the posterior cortex may be affected; the cataract may undergo partial involution, may remain stationary, or may advance to complete opacification of the lens. Various hypotheses have been advanced to account for its production.

Some believe that the lightning stroke causes a coagulation of albumin outside the lens fibres, and that the opacity may undergo involution when the albumin becomes redissolved (*Silex*). Others have ascribed it to the effect of heat.

These theories are improbable because the cataract does not appear immediately after a stroke of lightning, but later, after a variable length of time. I agree with *Hess* that a lens taken from an animal's eye cannot be rendered opaque by numerous shocks from a *Leyden* jar.

Vossius believed that a recurrent iridocyclitis after the shock may be the cause of the cataract, but such an iridocyclitis is not always present.

Knies assumed that the contraction of the ciliary muscle, induced by the stroke of lightning, dislocates the lens fibres and so causes the opacity, but this is incorrect, because we know today that the strongest contraction of the ciliary muscle can only relax the zonula and can produce no direct mechanical effect upon the lens. The view has also been expressed that the ultraviolet rays may be the cause of this form of cataract.

Hess determined the origin of this form of cataract experimentally. If a spark from a *Leyden* jar is allowed to strike the head of a rabbit in the region of the eye, a swelling of one side of the head, chemosis, cloudiness of the cornea and an opacity of the lens can be seen two days later. In such an experimentally produced cataract a fibrinous exudate is excreted into the anterior chamber, and an enormous hyperæmia of the ciliary body develops where the effect of the spark was most pronounced. After a few hours a delicate circular opacity generally appears at the equator, to which opacities of the posterior capsule associate themselves and spread toward the posterior pole. Opacities at the anterior pole, in the pupil, become visible later in most of the experiments. In the course of a few weeks the opacity of the lens either becomes total, or undergoes involution leaving an anterior polar cataract. This result depends on the degree of effect produced and on the individuality of the animal.

Hess has shown that in this form of cataract, just as in massage cataract, the first change consists of a destruction of the epithelium of the anterior capsule. In these cells are signs of degeneration, and fluid is collected between them and the lens fibres. The appearance of vacuoles in the neighboring lens fibres indicates that they also are undergoing disintegration.

The cause of cataract produced by lightning is therefore the death of the epithelium of the lens occasioned by the electric shock. Although this may not be purely a mechanical degeneration, yet it should be classed with the other forms of cataract in this group.

C. Opacities of the Lens Caused by Poisons

In 1886 *Bouchard* discovered that cataracts could be produced in rabbits by overfeeding them with naphthalin. Naphthalin dissolved in liquid paraffin is introduced into their stomachs by means of a probang.

The *naphthalinic cataract* begins with minute, perfectly clear radii which start at the equator, just beneath the capsule, and extend toward the poles of the lens, usually in the anterior cortex, less often in the posterior. They may be seen to sparkle when the mirror is rotated. They are clefts between the lamellæ of the lens which are filled with clear fluid, and have been compared quite appositely to cracks in clear ice. They may pass away completely when the overfeeding with naphthalin is discontinued, or the poisoning of the animal is not sufficiently grave, without going on to true opacity of the lens. If on the contrary the process is continued, opacities develop in various ways. The radii may be replaced by gray sectors, produced by the clouding of the clear fluid through the disintegration of the adjoining lens fibres and the escape of their constituents.

In other cases a gray circle is formed on the posterior surface of the lens near the equator. Not infrequently the posterior suture of the rabbit's lens can be seen distinctly as a gray stripe. Irregular spots and opacities of many forms may appear. Even in such cases an extensive involution of the lenticular disease is still possible. In the final stage the entire lens is opaque, and its cortex is of a uniform bluish white and gray appearance.

The origin of this disease of the lens has been subjected to a most rigid scrutiny, and the view that has finally been accepted is that the condition is one of poisoning of the lens.

Panas thought there was a connection between it and the retinitis excited by the naphthalin, but *Hess* has shown that the changes in the lens precede those in the retina, and that retinitis may be wholly absent, even when the cataract has existed for a long time.

According to *Magnus* and *Peters* the first symptom of naphthalinic cataract is a shrinkage of the lens, ascribed by *Peters* to disease of the epithelium of the ciliary body induced by the action of naphthalin, which results in an increase of the salt in the aqueous, which in turn causes a shrinkage of the nucleus of the lens by disturbance of osmosis. He arrived at this conclusion through determinations of the conductibility of the aqueous, but *Hess* found that the anterior chamber is shallower at the commencement of a naphthalinic cataract and that therefore the lens must have increased in volume. *Salffner* then showed that a few hours after the stuffing with naphthalin, when no changes in the lens can be recognized with the ophthalmoscope, the lens is always increased in weight and volume and not shrunken. When the first visible opacities appear the increase of weight and volume of the lens amounts to more than 16%.

The first symptom of naphthalinic cataract is the inability of the epithelium of the lens to prevent the entrance of water between the cells and fibres. At the same time the connection of the epithelium with the capsule becomes looser and undergoes still further disintegration, which leads to a rapid degeneration of the lens fibres. The lens reacts with processes of proliferation. Numerous mitoses are met with in connection with the destruction of the epithelium, and the posterior capsule becomes covered by a layer of pseudoepithelium during the first weeks of the development of the cataract. All of this indicates that the disease is produced by a poisoning of the lens.

The naphthalin itself is not the cause of the cataract. Animal lenses remain perfectly transparent even in a concentrated solution of naphthalin. The naphthalin must be given by mouth to produce a cataract, when administered in any other way it has no effect on the lens. Hence it must be supposed either that a product is derived from naphthalin in its passage through the intestinal tract, which is resorbed into the blood and is able to injure the lens, or that other substances are formed by the poisoning of the body which affect the lens. What these may be we do not know; they have not yet been detected either in the intraocular fluid or in the blood. We know only this, that lenses absorb water more quickly in the serum of animals that have been poisoned with naphthalin than in normal serum.

Occasionally cases of human cataract have been observed after the ingestion of naphthalin and naphthol, but the importance of naphthalinic cataract is chiefly due to the fact that of all experimental forms of cataract it is the one which presents most closely the kind of lenticular opacity met with in the commonest spontaneous form of human senile cataract. When the cortical layers of the human lens become spontaneously diseased, disintegrated and opaque, the condition is not like that produced by cold, heat, or mechanical degeneration of the epithelium of the capsule. The naphthalinic cataract shows us where to look for the causes of the human disease.

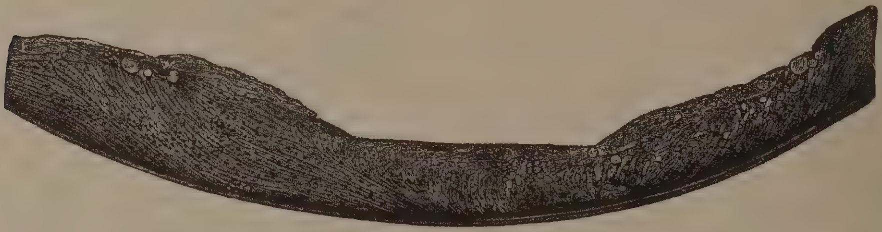


FIG. 45.—Pseudoepithelial Layer on the Posterior Capsule in Naphthalinic Cataract, after *Hess.*

Another poison which is able to cause cataract, when applied directly to the cornea, is the poison of bees. Now and then bee stings of the cornea are met with, and this peculiar injury is followed by a lenticular opacity confined to the most anterior layers of the cortex in the region of the pupil. This has been studied experimentally by *Huwald.* If an animal is stung by a bee in the centre of its cornea a minute opacity is formed in the pupil just beneath the anterior capsule. This spreads at first and then undergoes involution in a few days, leaving only a small anterior capsular cataract in the centre of the pupil. Anatomical study has shown that an irregular proliferation of several layers of capsular epithelium takes place. It is necessary that the poison be carried by the sting directly into the anterior chamber. Here again it is evident that we have to deal with a toxic action upon the epithelium and fibres of the lens in which specific affinities between the toxin and the substance of the lens play a part.

65. Lamellar Cataract and Its Treatment

Human cataracts may be divided into two large groups: the acquired and the congenital. The commonest form of congenital cataract is the cataracta zonularis, or lamellar cataract, which is illustrated by the present case.

A round, gray opacity, surrounded by a transparent marginal portion, may be seen by oblique illumination in the lens of each eye back of the plane of the pupil. The opacity is greatest at its margin, while its intensity decreases toward the centre, so that the latter seems to be more transparent. From this symptom we conclude that the nucleus is clear and that this is not a so called central cataract. After the pupil has been dilated with homatropin the eye may be examined with the ophthalmoscope, when the cataract appears as a central, dark disc surrounded by transparent and normally illuminated peripheral portions of the lens. It can also be perceived that the centre is less opaque than the margin of the opacity. Radiating dentations directed toward the periphery of the lens project from the sharply defined margin of the cataract.

With *Zeiss*' loupe an innumerable quantity of minute points, as well as larger gray white clouds, are to be seen in the opacity, which has hitherto appeared uniformly gray. The radiating, gray white dentations at the margin have a peculiar form. Part of them very distinctly have two legs, one belonging to the anterior, the other to the posterior layer of opacity. These opacities ride, as it were, on the margin of the lamellar cataract and have therefore been termed "riders." In some cases these riders lie more peripherally from the margin of the cataract in the otherwise clear layers of the lens. They are indicative of the commencement of a new layer of opacity. There are cases of lamellar cataract in which a second and a third layer of opacity have formed very distinctly about the principal one, between which are clear, or more correctly, less cloudy zones. In the great majority of cases congenital lamellar cataract is bilateral, and about uniformly developed in each eye.

Cases of lamellar cataract in only one eye have been described by *Davidson*, *Schirmer*, and *Hess*, and unequal development of the cataract in the two eyes has been met with. Cases also of so called rudimentary lamellar cataract. in which only a small part of the perinuclear portion was opaque, have been described by *Purtscher* and *Czermak*. Finally it must be mentioned that unilateral lamellar opacities may be acquired in the lens as the result of many diseases of the eye; such a condition has been met with after iritis by *Graefe*, after keratitis with perforation of the cornea by *Becker*, in leucoma adhærens after blennorrhœa by *Schirmer* and *Collins*, after injuries by *Hess* and others.

The size of the lamellar cataract may fluctuate materially; in this case it measures between 4 and 5 mm, others may measure from 2 to 8 mm across. The size of the opacity is important, for the impairment of vision depends greatly on its extent, as well as on its density. As a rule they are large enough to fill the pupil, and yet it is not a rare occurrence to meet with eyes containing lamellar cataracts in which the vision is nearly or quite normal. Most cases suffer a considerable loss of vision, which may be referred to the opacity, or to an imperfect function of the nervous apparatus. The presence of the latter is proved when good vision is not obtained after a faultless and early removal of the lamellar cataract. Nystagmus is not an infrequent accompaniment of these cases.

As soon as we have made the diagnosis of lamellar cataract our first duty is to determine the refraction and, if the patient is old enough, to test the function of the eye. It has been remarked frequently that eyes with lamellar cataract are almost always more or less nearsighted. The patient brings the print close to his nonatropinized eye when he reads, but this does not indicate positively that he has myopia. On the contrary the subjective test shows that he has a considerable degree of hypermetropia. The subjective test of the refraction is often insufficient to tell us more than this in such cases, especially in little children, so we are obliged to depend on objective methods, like skiascopy. Still clinical experience has proved that myopia is not rare in cataracta zonularis. *Schliep* found myopia in 22% of such cases, *Hess* in 11%. When myopia is present the ophthalmoscopic signs of elongation of the axis of the eyeball must be looked for; if they are absent it may be supposed that the myopia is due to changes in the lens itself.

Another point in the functional testing of such an eye is the determination of its power of accommodation. This is difficult when the vision is too poor to enable us to obtain accurate results. Such patients not infrequently have a sort of stenopæic vision, in which the rays of light dominate that enter between the margin of the pupil and the margin of the cataract. This likewise interferes with the accurate measurement of the accommodation. Yet there is no doubt that the accommodation of young patients with lamellar cataract is quite analogous to the normal. The cortical layers of the lens, in which the change of form takes place in accommodation, are normal in lamellar cataract.

Therapeusis depends on the result of this test of function. If the vision, perhaps after correction with glasses, is such that the patient can follow his calling, we leave the cataract alone. This may be done the more readily because the extent of a lamellar cataract usually remains stationary, although rare cases occur in which the intensity of the opacity increases.

In addition to the correction with glasses much improvement can be obtained in the vision of some patients by dilatation of the pupil with atropin, when the cataract is very small, sharply defined and so dense that the diffused light that passes through it does not interfere with vision. Such measures suffice for many years with some patients. Such a dilatation of the pupil takes the place of an operative form of treatment.

We have at our disposal two operative procedures for the treatment of lamellar cataract: optic iridectomy and removal of the lens. Both have their great advantages and disadvantages, and we must choose conscientiously which to advise in any particular case.

In an optic iridectomy a narrow, peripheral, transparent segment of the margin of the lens is made accessible to the entrance of light. This operation has the advantage that it preserves the accommodation of the patient so that he can use the eye for both far and near vision. In order to determine in any case whether it will be to the advantage of the patient to have an iridectomy performed the vision is tested first with a normal pupil, and again with the pupil dilated and a stenopæic slit employed, which corresponds in size and form to the coloboma which is contemplated, and the results thus obtained are compared.

The disadvantages of iridectomy are that the pupil is no longer round, and that the peripheral portions of the lens, which are not so uniformly curved, must be used for vision, so that the retinal images are not sharply defined and a considerable degree of dazzling may be the permanent result. Hence the operation is indicated only when the opacity of the cataract is sufficiently dense to prevent disturbance by diffusion of light, and when the peripheral zone of transparent lens is sufficiently broad.

The second operation is removal of the cataractous lens. The disadvantage of this operation is that the eye is made very hypermetropic, and loses its power of accommodation, so that the patient is compelled to wear different glasses for distinct distant and near vision.

On the other hand the advantages are that the pupil remains round and that the visual result is usually much better. The latter advantage in particular renders this procedure preferable whenever an operation must be performed.

The lens may be removed in three ways: Discission is performed in young children and in patients who may be expected to be unruly during and after the operation. We open the anterior capsule with a discission needle and thus intentionally produce a traumatic cataract. The lens will swell and become fully opaque in a few days, or sooner. The pupil is kept dilated with atropin and a close watch is kept over the tension of the eye. If the swelling is rapid and the tension becomes elevated, the lens may be evacuated through a paracentesis of the anterior chamber. In suitable cases the complete resorption of the lens may be awaited. Then it has to be decided whether an operation for secondary cataract will be required later.

In intelligent children an attempt may be made to remove the lamellar cataract, together with the adherent masses of cortical, directly from the eye through a section made in the limbus with a broad keratome, and after a broad opening has been made in the anterior capsule. If the technique has been mastered this operation succeeds very well and saves the patient considerable time. It may be sufficient alone, or a subsequent operation for secondary cataract may be needed.

If the operation for lamellar cataract needs to be performed on a patient who has arrived at a period of life when a marked sclerosis of the lens has taken place, the extraction should be made by means of the flap operation with preservation of a round pupil.

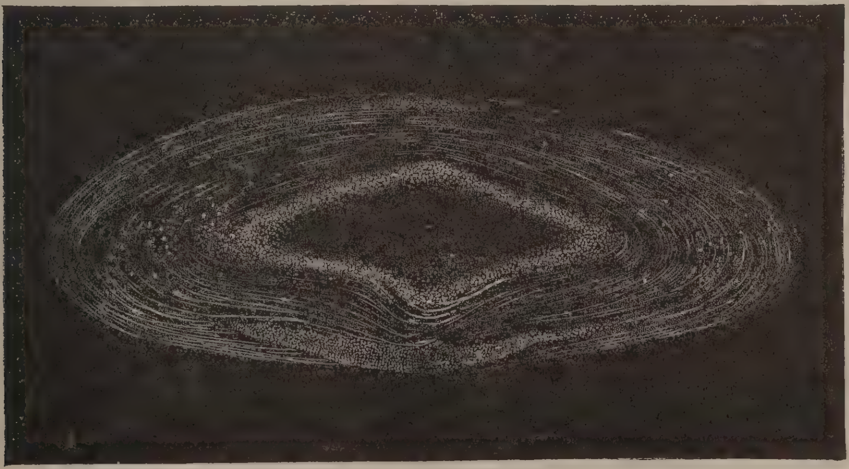


FIG. 46.—Lamellar Cataract, after *Hess*.

This peculiar form of cataract has always awaked the interest of ophthalmologists, and many anatomical researches have been made by *Deutschmann*, *Beselin*, *Schirmer*, *Hess*, *Peters*, *Treacher Collins*, *Meyer*, *Heinrich* and others. If we examine a section with a low power of amplification the zone of cataract between the nucleus and the cortex can be perceived immediately, for the zonular cataract is characterized by the appearance of a zone of degeneration between the central and peripheral portions of the lens, as its name implies. With a stronger amplification an extremely large number of very minute, round, or irregularly shaped foci of degeneration may be seen in this zone. These foci of degeneration may appear peculiarly laminated. *Hess* was unable to establish relations between them and the lamellæ by equatorial section. The nucleus also, which appears clinically to be normal, may be involved and contain in much smaller numbers such little foci or "drops." This involvement of the nucleus is not without importance to the full comprehension of the clinical picture. *Hess*, *Axenfeld*, *Baas* and others have repeatedly described the finding of globules and crystals in lamellar cataract. *Schirmer* has proved, by sections of freshly extracted cataracts of this nature, that the numerous little foci in the zone of opacity are not artificial products, or post mortal signs.

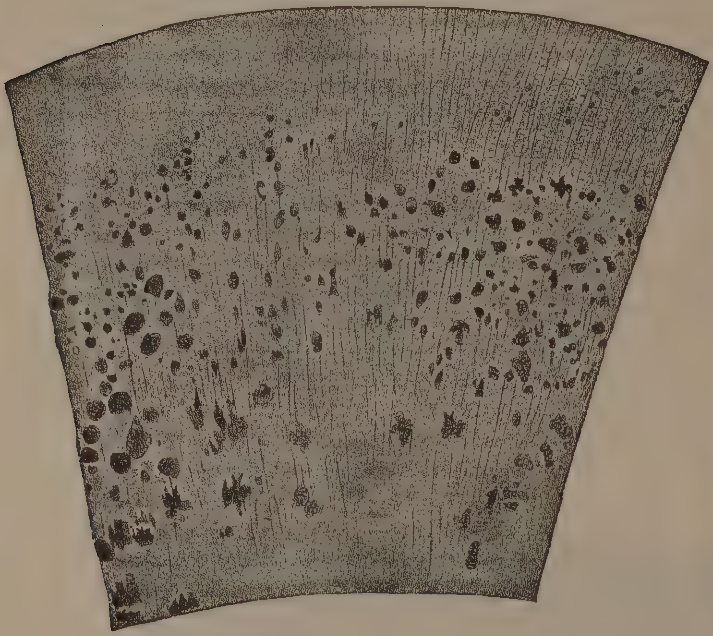


FIG. 47*a*.

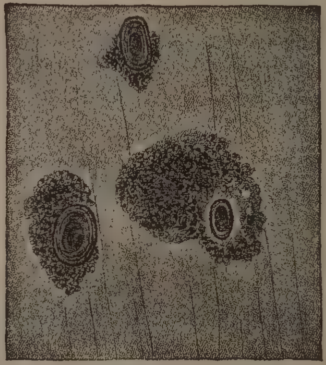


FIG. 47*b*.

Foci of Degeneration in Lamellar Cataract, after *Hess*.

No contraction of the nucleus, which has been assumed to exist and to be connected with the genesis, can be perceived. The size of the lens is normal and the condition of the nucleus in a section while fresh is the same as that of a normal lens of the same age, while its fibres prove on microscopical examination to be unchanged. Finally a word in regard to the nature of the so called riders. *Schirmer* and *O. Meyer* conceived them to be cleft formations caused by the supposed contraction of the nucleus, but *Hess* found that in his cases they were formed in the same way as the opaque zone itself.

Lamellar cataract is of interest not only to the specialist, but to the general practitioner. The nature of its origin is still enveloped in darkness. According to one theory, first voiced by *Becker*, cataracta zonularis is usually congenital. Other writers, like *Arlt* and *Groenouw*, think that it develops as a rule during the first year of life. A third group claims that it can develop just as well before as after birth. It has been detected immediately after birth by *Hosch*. However this may be, it is the duty of the physician to devote his attention not only to the eye, but also to the well being of the patient.

This condition is met with very often in families in which abnormalities of the eye are hereditary, and numerous cases have been reported in which congenital cataract was observed to appear through several generations.

It has also been noticed that very many patients with lamellar cataract suffer from convulsions during infancy, do not develop well mentally and have anomalies of the skull as well as peculiar malformations of the teeth. *Arlt* pointed out that lamellar cataract is met with particularly in children who have suffered from convulsions.

The teeth malformed are particularly the permanent upper incisors, eye teeth and first molars, which present horizontal ridges and grooves in the enamel and, in bad cases, may be worn away to little stumps. Both the teeth and the lens are epithelial formations and, according to *Horner*, such epithelial formations are apt to be attacked in rickets in addition to the bones.

An etiological connection has been assigned to rickets for convulsions in childhood, as well as lamellar cataract, by quite a number of authors, especially by *Horner* on the ground of exhaustive statistics. Rhachitis was found to be present by *Ruehr* in 88% of 153 cases of lamellar cataract, by *Schliep* in 53% of 170 cases. Probably the actuating noxious quality of the disease, the nature of which is still unknown, induces the morbid changes in those layers of the lens that are present, or in process of formation at a certain time. If the general disease passes away normal lenticular layers may afterward be formed. This hypothesis renders comprehensible the peculiar localization of the opacities in lamellar cataract between the nucleus and the cortex.

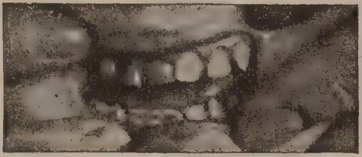


FIG. 48.—Rachitic Teeth.

We also know that lamellar cataract is scarcely to be met with in countries in which rickets is rare. Although this coincidence of the two diseases is indicative of some relation between this metabolic disease and the abnormality of the lens, yet it has not been proven that rickets is the true cause of lamellar cataract. Much more thorough investigation is needed. Some authors, such as *Peters* and *Kirchhoff*, incline to make tetany, rather than rickets, responsible for the development of lamellar cataract, and the ascription of the dental changes to rickets has also been disputed. There is no doubt that many peculiarities in the pathogenesis of lamellar cataract still remain unexplained, such as the involvement of the nucleus, the displacement of the nucleus near the posterior pole of the lens, which I have met with several times, and the unmistakable relation of lamellar to other congenital forms of cataract, such as the central and the spindle shaped. In relation to this *Hess* has met with interesting disturbances in the separation of the lens vesicle from the epiblast in the embryonal lens of a young chicken, which suggest the idea that such disturbances may at least take part in the causation of congenital lamellar cataract.

The prognosis of the operation for the removal of lamellar cataract is good. In most cases a not inconsiderable improvement of vision is secured.

66. Rarer Forms of Congenital and Acquired Cataract

The most important and the most frequent forms of cataract have now been considered, but others are met with, though they are comparatively rare. Although the lens seems to be constructed so simply, yet the most manifold clinical pictures appear in it. The reason for this variety is that the lens is affected very easily by local diseases of the eye. Constitutional causes may also be the basis of the rarer forms of cataract. Very little is known as yet in regard to their etiology. Part of them are congenital, part acquired.

Cataracta Polaris Anterior

I exhibit here a child who has in the centre of the pupil of each eye, at the anterior pole of the lens, a small, sharply defined, intensely white opacity. Elsewhere the lens is perfectly transparent and no other morbid changes are to be found in the eye.

Anterior polar cataract is generally stationary. When it is as small as it is in this case it causes very insignificant disturbances of vision, or none at all; but when it is larger, so as to cover a great part of the pupil, much visual disturbance is complained of, especially nyctalopia. During the day, when the pupil is contracted by the bright light, the patient sees badly, but in the twilight, when the pupil is somewhat dilated, his vision is better, because the rays of light gain a freer entrance beside the opacity. Optic iridectomy is to be considered in such cases, after it has been demonstrated that the vision is actually improved by the dilatation of the pupil.

Not infrequently anterior polar cataract is found associated with other congenital forms in the same lens, such as lamellar cataract, and central cataract. In such cases extraction of the lens is to be considered.

We try to explain the origin of congenital anterior polar cataract by supposing that, in consequence of a derangement of development, the separation of the lens took place irregularly and left an opacity at the place of separation.

Anatomically, we find that the anterior polar cataract is produced by a circumscribed proliferation of the epithelium of the capsule.

Anterior polar cataract may also be acquired. Here is a child a year old, who had blennorrhœa neonatorum. One eye is intact. In the other is a central leucoma of the cornea and directly opposite it in the pupil is a sharply defined, white opacity at the anterior pole of the lens.

The origin of this polar cataract can be read from the eye itself: it was caused by the ulcer that formerly existed in the cornea. When a central ulcer of the cornea perforates the aqueous escapes, the lens moves forward and comes into contact with the ulcer. The partly mechanical, partly inflammatory irritation thus produced excites a proliferation of the epithelium at the place of contact on the capsule. The ulcer heals, the anterior chamber is reformed, but a leucoma of the cornea and an anterior polar cataract remain.

Such an etiology must be borne in mind even when the cornea is perfectly transparent, for the cornea is often capable of clearing up wonderfully in children, and an extremely delicate opacity must always be searched for.

Occasionally such an anterior polar cataract is united to the central corneal cicatrix by a thread of connective tissue. In such cases the ulcer was joined to the lens by a mass of exudate, which became organized and was drawn out into a thread after the reformation of the anterior chamber. This thread may break, or it may remain permanently.

An anterior polar cataract caused in this manner may not form simply a flat opacity; it may protrude more or less into the anterior chamber. It is then known as a cataracta pyramidalis, which is always acquired.

Anterior polar cataracts have also been observed to develop when the perforating ulcers were not central, but peripheral, from the irritation exercised upon the epithelium, and also to develop when the ulcers did not perforate. They may likewise be produced by suppuration in the anterior segment of the globe, for *Wagenmann* saw a case of fresh iridocyclitis in which there was a proliferation on the capsule where pus cells had come into contact with it. I have repeatedly seen a polar cataract develop after ulcus serpens in adults.

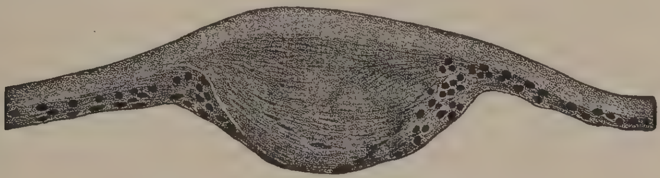


FIG. 49.—Capsular Cataract, after *Hess.*

Acquired and congenital anterior polar cataracts exhibit analogous histological changes. The epithelial cells at the anterior pole are first destroyed, and then a proliferation of the capsular epithelium takes place which results in a capsular cataract.

Cataracta Polaris Posterior

The differentiation between the congenital and the acquired forms of posterior polar cataract is important, because the latter is often a symptom of disease elsewhere in the fundus of the eye. For this reason the fundus should be investigated as soon as an opacity about the posterior pole of the lens is detected.

Congenital posterior polar cataract on the contrary is not dependent on disease of the fundus. At the posterior pole of the lens is a sharply defined dark point, which is shown to be deeply situated by the fact that its parallactic movement is in the opposite direction to that in which the eye of the patient is moved. These congenital posterior polar cataracts are divided into two forms: cataracta polaris posterior spuria and vera.

If by the term cataract is understood a degenerative process within the lenticular capsule, a posterior polar cataract will be termed vera when the lens fibres at the posterior pole are morbidly changed. Such congenital opacities often occur coincidently with lamellar cataract and central cataract, and may then be referred to similar causes.

In the other congenital form the opacity lies on, not within, the capsule at the posterior pole of the lens. During fœtal life the hyaloid artery passes through the vitreous to the lens, and when its involution is incomplete some tissue remains on the posterior capsule and forms a posterior polar cataract. In these cases the artery may be found sometimes to be persistent, or to be represented by a solid cord that extends from the posterior capsule to the papilla, or by only a floating filament, or the only trace may be a little round opacity on the capsule. If the eye is otherwise normal the vera can hardly be distinguished from the spuria, unless remains of the hyaloid artery are to be seen distinctly, but if the condition is met with in a microphthalmic eye that has other anomalies of development the diagnosis is made more easily. Such opacities have also been observed in connection with lenticonus.

Acquired posterior polar cataract is associated particularly with two diseases of the fundus: pigment degeneration of the retina (retinitis pigmentosa), and chorioiditis.

In pigment degeneration of the retina a punctate opacity is apt to develop at the posterior pole of the lens, to which some radiating opacities are gradually added, but it is characteristic of this form of cataract that it has no tendency to change into a total opacity of the lens. We do not know how this disturbance in the nutrition of the lens takes place, but it may be mentioned that *Wagenmann* saw posterior polar cataract develop after partial section of the ciliary vessels of an animal.

The anatomical basis of a polar cataract from retinitis pigmentosa is a proliferation of the capsular epithelium from the equator over tracts of the posterior capsule, with disintegration of fibres, cleft formations and little hollow spaces at the posterior pole itself.

Another kind of posterior polar cataract, not infrequently met with in chorioiditis, appears in the form of rosettes, or stars, tends to spread and sometimes involves the entire posterior cortex after a short time. The prognosis of its removal is bad, because the vision is seriously compromised by the disease in the fundus.

Posterior polar cataracts are also met with in other diseases, such as the uric acid diathesis, and stellate opacities at the posterior pole may follow injuries, either with or without demonstrable lesions of the capsule.

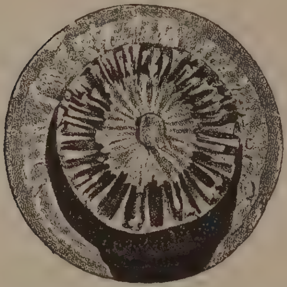


FIG. 50.—Central Cataract, after *Hess*.

Cataracta Centralis

Associated with lamellar opacities in the lens of this small child may be seen a white opacity in and somewhat behind the centre, which passes over into peripheral linear and punctate opacities. Central cataract can be differentiated anatomically from lamellar only by the fact that the disintegration of the nucleus is more advanced than is usual in zonular cataract.

Cataracta Fusiformis

Cataracta fusiformis is one of the congenital forms. The characteristic of a well marked case is an opacity that resembles a tube, or an ampulla, which extends

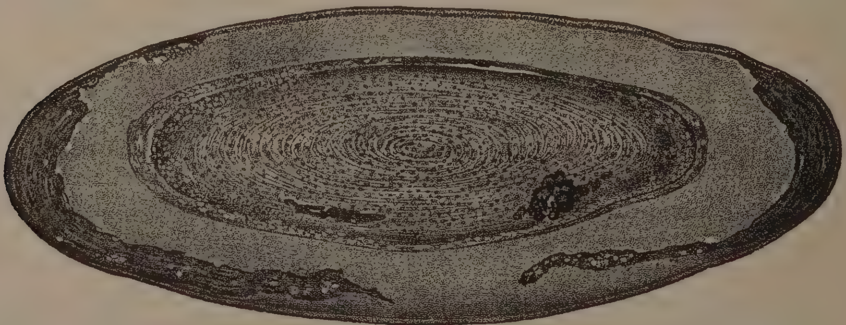


FIG. 51.—Congenital Total Cataract, after *Hess*.

through the middle of the lens from the anterior to the posterior pole. With this form is also grouped the spindle shaped cataract, in which such a union exists only between the anterior pole and the nucleus. It is caused probably by a disturbance in the separation of the lenticular vesicle.

The lens of a child a few weeks old may be totally and uniformly white, and present no other peculiarities that can be perceived clinically. This is a congenital total cataract. As the opacity is close beneath the capsule it is evident that all the lens fibres formed at the time of birth have disintegrated.

Part of these congenital total cataracts are far advanced lamellar cataracts, but it is possible that in others some nutritive disturbance attacked the cortical layer during fœtal life and produced a total opacity of the lens.

It has also been observed that a lens which was transparent at birth may become opaque from masses of connective tissue about a persistent hyaloid artery.

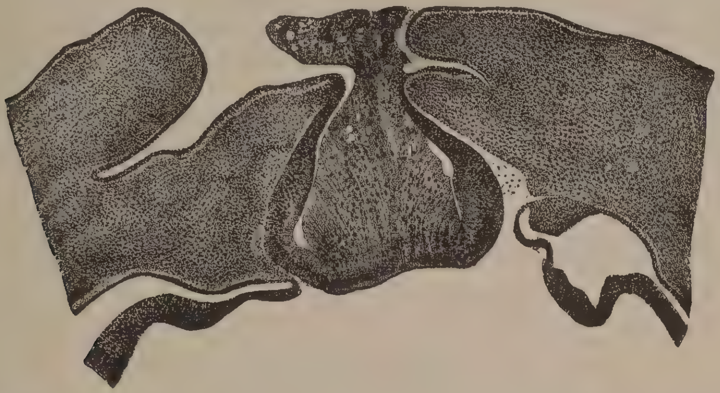


FIG. 52.—Disturbance in the Separation of the Lens Vesicle, after *Hess.*

Children are very often seen with congenital cataracts which have become shrunken. Soft, opaque lens matter may naturally undergo further changes in cataracts that are congenital, as well as in those of adults. Usually they appear flat and chalky white. The depth of the anterior chamber and the tremulousness of the iris indicate their shrunken condition. Such a cataract may be the final stage of a congenital total cataract, an advanced zonular, or it may be that a fœtal rupture of the posterior capsule has resulted in a gradual opacification of the entire lens, or possibly it is a cataract caused by an intrauterine inflammation. In this last form syphilis is a prominent agent; our attention is being drawn more and more to the fact that such cataracts may follow a syphilitic intrauterine uveitis.

67. The Development of the Lens and Its Congenital Abnormalities of Form and Position

The eye originates from the wall of the primary prosencephalon, from which the primary optic vesicle bulges out on each side of the median line. This primary optic vesicle is connected with the cerebral vesicle by a pedicle which later becomes the optic nerve. The primary optic vesicle is covered by ectoderm. After a very short time the secondary optic vesicle develops from the primary optic vesicle by a distal and ventral invagination of its wall, or, to speak more correctly, by a growth forward of the margin over the base of the optic vesicle. Thus a double-walled structure is obtained that presents ventrally the fœtal ocular cleft, which is continued to the pedicle of the optic vesicle. At the distal opening of the optic vesicle is the lens.

The ectoderm on the summit of the primary optic vesicle begins to grow more vigorously and forms at first a pouch directed toward the primary optic vesicle, connected with the surface by a little pedicle which is finally constricted off, so that the ectoderm has grown together over it at the end of the first month.

Thus the lens is an offspring of the outer germinal layer, an epithelial formation, and consists at first of a hollow vesicle. Then the cells of the proximal wall of the lens vesicle develop into lens fibres, while from the cells of the distal wall comes the capsular epithelium, until the lens has become a solid structure. Fibres continue to be formed throughout life from the equatorial capsular epithelium.

It is important to remember, in connection with its congenital abnormalities, that the lens during fœtal life is enveloped in a vascular capsule, the vessels of which are connected with the hyaloid artery and with it normally undergo involution.

The retina comes from one layer of the secondary optic vesicle. Its anterior margin reduces itself to a single layer of cylindric cells and forms the pars ciliaris retinæ, from the cells of which the fibres of the zonule are developed.

In such a complicated embryology of the lens it is conceivable that disturbances may occur in its development, and that these may result in abnormalities in either its form, or position. In fact we do meet with abnormalities in both the form and the position of the lens.

A. Congenital Abnormalities of Position

Ectopia Lentis

This patient states that the vision of both of his eyes has been bad since childhood. The most striking point in the appearance of his eyes is that the pupils are not central, but are displaced upward. After one of the pupils has been dilated with cocain it can be seen by oblique illumination to be divided by a circular edge, the margin of the lens, into two portions, one slightly gray, the other deep black. The gray appearance is the physiological reflex of the lens, and we have before us a pupil one portion of which is occupied by the lens while the other portion is aphakic.

The lens may be seen with the ophthalmoscope to be perfectly transparent, except at its edge where it appears to be black. The edge is sharply defined at the margin, less so toward the centre.

By focal illumination again the anterior chamber is seen to be unevenly deep, shallower in the portion in which the lens is present. The iris is tremulous, especially in the aphakic portion of the pupil.

The great difference in the refraction of the two portions of the pupil is revealed by skiascopic movements of the mirror. Myopia is often present in the one portion, while hypermetropia exists in the aphakic portion because the curvature of the lens when thus dislocated is not uniform. This may result in monocular diplopia, or, when the ectopia is bilateral, in polyopsia.

In a word the same symptoms are present in congenital dislocation of the lens as in acquired dislocation, but for practical reasons we designate the acquired dislocation as subluxation, or luxation, the congenital as ectopia. It must be understood, however, that an ectopia may become a luxation, either by traumatism, or spontaneously. This distinction between the two forms of dislocation is justified because ectopia is marked by many peculiarities. It differs from luxation in being almost always bilateral, and in that the lens usually is displaced symmetrically in the same direction in the two eyes, upward, upward and outward, or upward and inward. Displacements in other directions and asymmetric dislocations are occasionally met with. The pupil is usually displaced in the same direction as the lens.

Heredity plays an important part in its etiology. The lens may remain clear for a long time, sometimes for life.

Therapeutic intervention is not necessary in most cases of ectopia. We simply try to obtain the best vision possible by means of suitable glasses. Extraction may be considered when the lenses become opaque, become luxated in consequence of increasing atrophy of the zonule, or when secondary glaucoma develops.

The origin of ectopia is of interest in connection with the embryology of the lens. It cannot be accidental that the ectopia is almost always upward.

A persistence of the hyaloid artery and of mesodermal tissue cords in the vitreous, which are connected with the closure of the secondary optic cleft, can be demonstrated anatomically in these cases. We imagine, with *Hess*, that such cords both interfere with the development of the zonula and drag the lens upward. Such cords may disappear so that they cannot be found later. We know that the connections between the ciliary body and the lens are formed in the fourth month. Such cords are usually found below, in connection with the fœtal ocular cleft.

There are cases of ectopia in which the lens is met with close to the head of the optic nerve and attached to it by mesodermal cords of connective tissue, which penetrated through the ocular cleft.

In cases of lenticular astigmatism and lenticular myopia a slight degree of ectopia should be thought of, as it may easily be overlooked. On the other hand, ectopia is not to be diagnosticated in every case in which the margin of the lens can be made visible in the pupil, because it may appear as a straight line in coloboma.

B. Congenital Abnormalities in the Form of the Lens

I. Lenticonus Posterior

This little girl has always seen badly with one eye. What appears to be an opacity far back of the level of the pupil can be seen by focal illumination. With the loupe it can be seen that the posterior surface of the lens is conical, but no true opacity can be made out by transmitted light. True opacities are met with in some cases of this nature. The ophthalmoscopic examination reveals nothing of interest.

On rotating the mirror peculiar alternating shadows flash up at the margin between the middle and the peripheral parts of the pupil. With a concave mirror the illuminated field in the centre of the pupil moves distinctly in the same direction as the movement of the mirror, while the movement is not as distinct in the peripheral parts. Hence we conclude that there is a fairly high degree of myopia in the centre of the pupil. Sometimes the difference in the refraction of the central and peripheral portions of the pupil amounts to more than 10 D. This resembles to a certain extent the changes met with in the lens with a double focus, which has consequently been called false lenticonus. But false lenticonus is an acquired anomaly of the nucleus of the lens, which is of normal form, met with in elderly people. True lenticonus is usually congenital, though it may occasionally be considered acquired. It is primarily a change in the *form* of the lens.

The distinctive characteristic of true lenticonus is that the lens is conical, either on its anterior or its posterior surface. We meet with lenticonus anterior and posterior, the latter more frequently.

The posterior lenticular image is much smaller where the posterior surface of the lens is most strongly curved than in its vicinity, and if the image is caused to move about by moving the light, it elongates in passing from one zone to another, and disappears in places.

From these symptoms it may be diagnosed that the lens has undergone an essential change of form in its posterior segment, that the portion of lens near the posterior pole has protruded toward the vitreous. The peculiar reflexes obtained have been compared to the image produced by a drop of oil on a lens.

It was not without purpose that attention was called to the fact that the hyaloid artery extends to the lens during fœtal life. *Hess* has frequently found remains of the hyaloid artery in these cases, and other developmental anomalies of the eye are often found associated with lenticonus.

This abnormality of the lens is met with not infrequently in the eyes of animals. *Hess* found anatomically that the posterior capsule in the vicinity of the posterior pole was very thin and, in almost all cases, greatly lacerated. It is always difficult to detect such a tear in the living person. When opacities are present we can suspect that such a laceration is there, and account in this manner for the irregularity of the lenticular images. We must assume, however, that lenticonus posterior is connected with disturbances in the involution of the hyaloid artery, which probably occasion lacerations of the capsule. The nucleus may then be displaced backward. The only therapeusis is to try to improve the vision with glasses, an attempt that is not always successful.

II. Coloboma Lentis

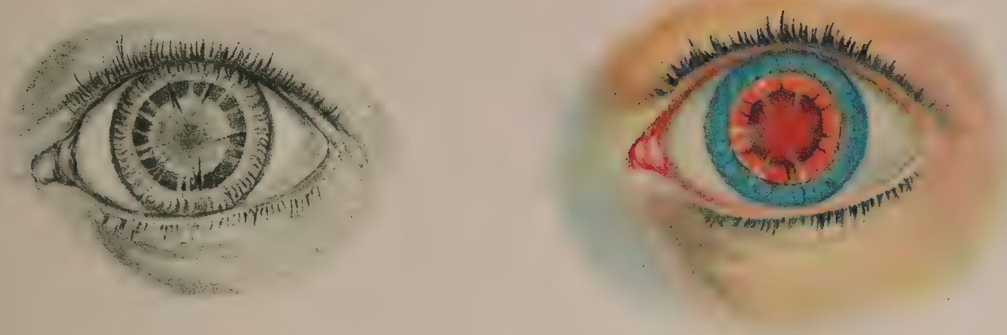
This patient has a congenital coloboma of the iris and chorioid, and in the coloboma of the iris may be seen a distinct, saddle shaped notch in the margin of the lens. Attention has already been called to the fact that the normal margin of the lens is not perfectly smooth, but such a notch as this is not physiological, it extends deeply into the substance of the lens. Frequently the margin forms a straight line instead of being circular.

The condition of the zonule may be studied with the ophthalmoscope. In this case it can be demonstrated distinctly in the region of the defect, but this is not true in all cases.

The effect of coloboma upon the vision depends primarily on whether it is associated with other abnormalities. In more than 50% of the cases coloboma of the lens is associated with coloboma of the membranes of the eye. The vision of such eyes is bad. When the coloboma is present in the lens alone it causes no visual trouble and is often discovered accidentally. Very large colobomata may give rise to an irregular lenticular astigmatism.

Three hypotheses have been advanced to account for its origin. I agree with *Hess* that certain vessels of the fœtal capsula lenticularis may remain longer than normal at the equator of the lens. If the lens grows rapidly such persistent vessels will press on its margin and lead to the formation of a coloboma at this place by mechanical inhibition of growth.

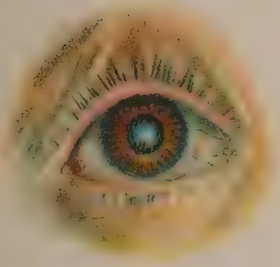
There are also great individual differences in the development of the mesodermal tissue which protrudes through the cleft of the optic vesicle into the vitreous



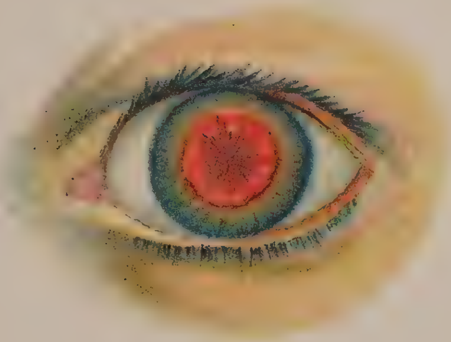
a Cataracta zonularis.

1 By direct light.

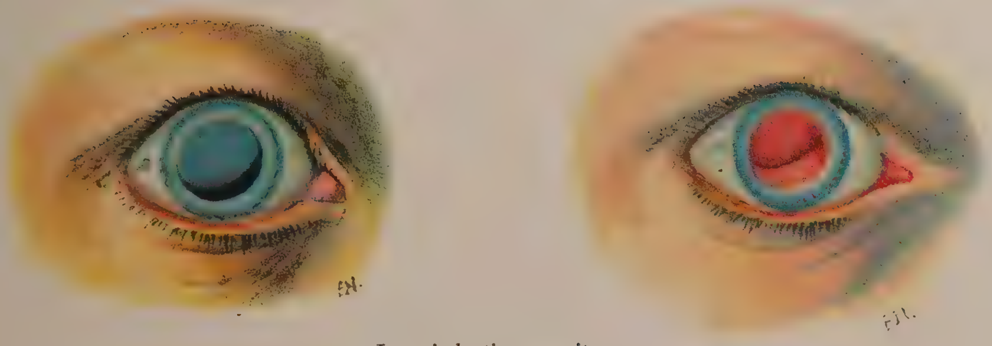
2 By oblique light.



b Cataracta polaris anterior.



c Posterior cortical cataract.



d Luxatio lentis congenita.

1 In oblique illumination.

2 In direct illumination.

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space, as has been shown by anatomical examinations of malformations of the eye. Such connective tissue cords in the vitreous may explain a part of the colobomata of the lens that are situated below.

The absence of the zonule in the region of the coloboma of the lens is also to be considered etiological. The margin of the lens may be abnormally curved at such places, or the lens may grow imperfectly. This kind of coloboma of the lens must always be shallow.

V

Diseases of the Eyelids

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68. Chalazion and Other Diseases of the Tarsus

This patient has a circumscribed swelling in the lid, the situation of which should be determined accurately.

When we study a section of the lid we find it to be composed of skin, tarsus, muscles, nerves and mucous membrane. The skin contains glands and minute lanugo hairs; beneath the subcutaneous tissue we trace the cut bundles of the orbicularis, beneath this lies the tarsus, which is composed of dense connective tissue. The tarsus harbors the *Meibomian* glands and in its upper part some mucous glands, the so called *Krause's* glands. Between the lower margin of the tarsus and the free margin of the lid lies the musculus subtarsalis, or *Horner's* muscle (musculus *Riolani*). The inner surface of the tarsus is covered by conjunctiva. In the anterior portion of the margin of the lid are to be seen the hair bulbs of the cilia, together with the mouths of the sweat glands (*Moll's* glands), and of the sebaceous glands (*Zeiss'* glands). The muscles present are 1, the levator of the upper lid, supplied by the oculomotorius; 2, the orbicularis, supplied by the facial. This muscle surrounds the palpebral fissure, and its deeper part, which is employed in gentle closure of the lids, is represented by the musculus subtarsalis; 3, the so called *Mueller's* muscle, a smooth muscle found in both the upper and the lower lid, supplied by the sympathetic.

The skin of the normal lid is very delicate and that of the upper lid is folded when the eye is opened. This folding is due to a division of the fibres of the levator, and is called the covering fold of the lid. The posterior lamella of the tendon of this muscle is inserted into the upper margin of the tarsus, while another lamella extends down its anterior surface and sends attachments to the skin. The skin passes into the mucous membrane at the free margin of the lid, where it forms a strip 2 mm broad, the intermarginal portion, the anterior edge of which is somewhat rounded off and presents the cilia jutting from it, with the mouths of the *Meibomian* glands in front of them, while the inner edge is sharp and forms almost a right angle. The margins of the lids approach and unite at their outer and inner ends to form the outer and inner canthi. The inner canthus is shaped like a horse shoe and contains the caruncle.

Where is the seat of the disease in this case? Palpation shows that the little tumor, about as large as a pea, does not lie in the skin which is movable over it, although in many cases it is adherent. It is roundish, nodular and elastic. We can move it together with the tarsus, to which it is intimately attached. After everting the lid we see a reddish gray, rather transparent place, corresponding to the site of the node, showing through the conjunctiva, which is red and swollen in the vicinity of the growth. The tumor lies in the tarsus. It has developed very gradually without pain or annoyance, except for the disfigurement it produces.

The tarsus consists of connective tissue with elastic fibres, not of cartilage, as it is often thoughtlessly said to be. An impression of its thickness and elasticity may be obtained by taking the lid of a closed, healthy eye between the thumb and forefinger. It contains the *Meibomian* and accessory *Krause's* glands. Hence a disease of the tarsus may be either an affection of the glands, or of the connective tissue substance of the tarsus itself.

I. Diseases of the Glands of the Lid

The function of the *Meibomian* glands, which lie close together, with the orifices of their emunctory ducts just in front of the inner edge of the lid, is to lubricate the margins of the lids. This function may be augmented, or diminished.

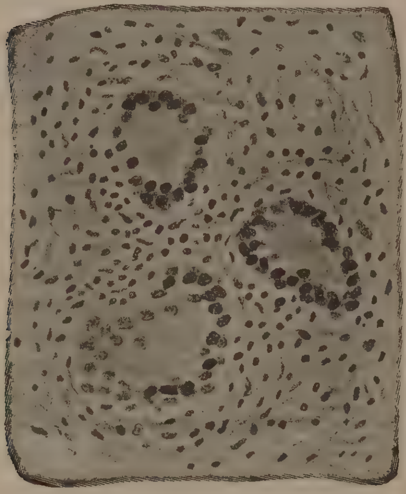

FIG. 53.—Giant Cells in a Chalazion.

In cases of hypersecretion of the *Meibomian* glands a whitish scum appears on the margins of the lids, at the canthi, and on the caruncle, which dries at night. This hypersecretion is often associated with seborrhœa of the margin of the lid. In other cases the glandular secretion is diminished. The glands must also suffer when the tarsus undergoes contraction in trachoma.

Our patient has a spontaneous, chronic, granulating inflammation of the *Meibomian* glands, which we designate *chalazion.*

The pathogenesis of this disease is still obscure, and its pathology must first be understood in order to appreciate the theories that have been advanced. The contents of a chalazion consist of a mucous, soft granulation tissue containing many round cells and very many giant cells which have an epithelioid character and contain numerous nuclei, usually arranged irregularly, more rarely about the margin. Dilatations of the preserved glandular alveoli, calcified masses of secretion, remains of dead epithelia and infiltration of the surrounding connective tissue of the tarsus are also to be seen. The condition is one of chronic folliculitis with development of a granulation tissue containing giant cells. *Virchow* thought chalazion to be a granulation tumor, while *Haab, de Wecker* and others believe it to be a retention cyst of the *Meibomian* glands. The great number of giant cells led *v. Baumgarten* and *Tangl* to think it a tuberculous granulation tissue, but it has been proved by very many inoculation experiments that chalazia do not contain tubercle bacilli, and I do not believe that they are in any way connected with tuberculosis. It can happen only in rare cases that tubercle bacilli may be carried metastatically into the connective tissue of the tarsus and excite a formation of granulation tissue in the glands. During experiments concerning endogenous tuberculosis of the eye *Stock* produced chalazionlike nodes in the upper lid. Other microorganisms have been taken to be the agents of chalazion. *Deyl* believed he had discovered the agent in the xerosis bacillus, but this was an error.

Deyl and *Hala* injected masses of xerosis bacilli beneath the skin and conjunctiva with the result that nodules were produced, but these reactions of the tissue to the injection of such masses of bacteria give no indication in regard to the infectious nature of xerosis bacilli, as *Bietti* and *Axenfeld* have pointed out. The agent of chalazion is still unknown, though the tendency of the disease to recur indicates an infectious etiology. Research among the protozoa may perhaps be more successful.

When left to itself a chalazion rarely remains stationary. Sometimes it becomes smaller because of atrophy of the granulation tissue. Usually it grows larger gradually until the proliferating granulation tissue breaks through the conjunctiva tarsi and protrudes through the perforation into the conjunctival space. A chalazion may be single, but frequently many are present in both the upper and lower lids. A number of adjoining *Meibomian* glands may become diseased and give rise to a flat, broad swelling, which is called a giant chalazion. The granulation tissue often exists only in the efferent ducts of the *Meibomian* glands and forms reddish gray nodules on the margin of the lid; chalazion marginale.

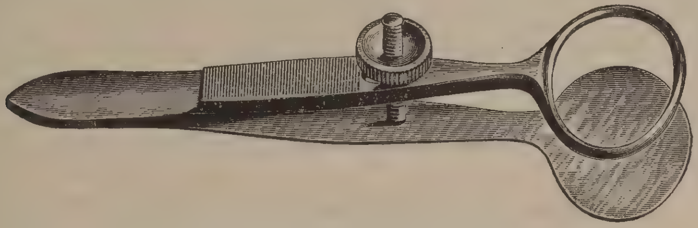

FIG. 54.—*Desmarre's* Chalazion Forceps.

To remove a chalazion some cocain is injected in its vicinity, the lid is everted and seized with chalazion forceps so as to render the tissue bloodless, the conjunctiva is incised parallel to the course of the *Meibomian* gland, and an attempt may then be made to dissect out the nodule with scissors and forceps. Usually, however, a part of the contents escapes. The cavity is then to be curetted thoroughly to prevent recurrence, after which a bandage should be applied for a day or two. If it is desired to extirpate the chalazion through the skin, a horizontal incision is made parallel to the margin of the lid down upon the tumor, and is later closed with sutures. In the diagnosis of a chalazion it is but rarely that a tuberculous or gummatous node needs to be considered.

Dilatations of the efferent ducts of the *Meibomian* glands are, on the contrary, frequently met with in the course of chronic conjunctivitis, and of diseases of the margins of the lids. Little whitish yellow spots may then be seen on the conjunctiva tarsi, over which the conjunctiva is somewhat arched and swollen. This condition is one of *occlusion of the efferent ducts* by masses of secretion that have undergone partial calcification. These formations must be looked for and removed when there is a constant feeling as of a foreign body in the eye during the course of a chronic conjunctivitis. *Mitvalsky* observed a colloid transformation of the contents of the efferent ducts, and hard pearls, as clear as glass, may be removed sometimes from the glands.

Cysts of the glands appear through the conjunctiva like transparent formations. Primary tumors, particularly adenomata, of the *Meibomian* glands are met with. *Krause's* glands, which lie in the posterior margin of the tarsus, may also be the seat of disease, and may become involved in inflammations of the conjunctiva. Cysts, and in rare cases adenomata, develop in them. As a rule, the morbid processes met with in the glands of the lid are chronic.

The acute purulent inflammation of the *Meibomian* glands, the so called *hordeolum internum,* forms a complete contrast to the chronic granulating inflammation met with in chalazion. In this condition a hard node likewise may be felt in the tarsus, but it is very painful and the symptoms of inflammation are tempestuous. The inflammatory swelling involves the skin of the lid as well as the conjunctiva tarsi and bulbi. The preauricular gland is enlarged and tender; the clinical picture is in a word that of an acute, deep abscess of the lid. The pus soon causes a yellow discoloration to appear on the conjunctiva tarsi, which is finally perforated and the abscess is evacuated into the conjunctival sac. This condition we call *hordeolum Meibomianum.* In many cases the conjunctiva secretes a larger quantity of sero purulent fluid. The suppuration is caused by the entrance of virulent staphylococci into the *Meibomian* glands through their efferent ducts. After the pus has been evacuated by a timely incision the inflammatory symptoms rapidly abate. Sometimes a granulation forms in the opening of the abscess cavity and very malignant cases may result in an extensive necrosis of the tarsal connective tissue. Such cases are spoken of as tarsitis necroticans.

II. Diseases in the Tarsal Connective Tissue

Isolated diseases of the connective tissue of the tarsus are rare. The tarsus may naturally be involved in many local inflammations of the skin and conjunctiva, such as erysipelas, grave infectious forms of conjunctivitis and trachoma, but in all such cases the primary disease forms the centre of interest, while that of the tarsus is secondary.

Other diseases of the tarsus are very rare. *Amyloid degeneration of the tarsal connective tissue* is sometimes met with as a fatty, gelatinous, waxlike, firm mass on its inner surface; whether it starts from the conjunctiva or the tarsus cannot be determined clinically. There are only two diseases of the tarsus that need to be differentiated from chalazion: tuberculosis and syphilis.

Tuberculosis of the tarsus is not often met with as an isolated condition. It is usually either a transplantation of a primary tuberculosis of the conjunctiva, when little nodules appear between the tarsus and the conjunctiva in connection with a tuberculous ulcer of the latter, or an extension of a tuberculosis of the skin. When the tarsus is primarily affected endogenously, circumscribed thickenings are formed, the disease extends to the glands and a diagnosis may not be possible except through a histological examination.

Syphilitic disease of the tarsus may occur either in the form of a diffuse gummatous infiltration, or of circumscribed nodes. When the infiltration is diffuse severe inflammatory symptoms, such as swelling of the lids, are present and the fatty tissue may show through the conjunctiva, the same as in chalazion. More often the gummatous nodes are circumscribed and hard. They are usually present in both lids, and the clinical picture may be similar to that of chalazion. These syphilitic diseases of the tarsal connective tissue may be met with in both hereditary and acquired syphilis and, although chalazion is the disease of the tarsus most frequently met with, they must be borne in mind in certain cases and treatment governed accordingly. Finally, it should be mentioned that a sarcoma or fibrosarcoma may occasionally originate primarily in the tarsus. Congenital entropion is indicative of a faulty development of the tarsus.

69. Of What Diseases Are Disturbances of the Circulation in the Skin of the Lid Indicative?

The lids of this young girl suddenly become œdematous at irregular intervals of several weeks without any demonstrable cause. The lids of both eyes are much swollen, their skin is of a pale, waxy yellow color. There are no signs of inflammation and no itching. The swelling lasts a few days and then disappears to return again in a few weeks. This is the so called recurrent œdema of the lids, which is also called *Quincke's* disease. It may appear alone as in this case, or it may be associated with œdema of the skin of the face, or of other parts of the body, or even of the mucous membrane. Its nature is not yet fully understood, but it is generally thought to be a vasomotor disturbance coming from the central nervous system. Treatment of this angioneurosis is limited to gentle massage of the lid and attention to the general condition. The prescription of drugs should be made dependent on the result of the examination of the blood.

The first question to be answered, when we are called upon to diagnose a disease of the lids, is whether the disease is located in the tarsus or in the skin. The most frequent symptoms in the skin of the lid are 1, disturbances of circulation; 2, inflammation. This case is an example of the former.

The principal circulatory disturbances to be considered are hemorrhages, arterial hyperæmia, hyperæmia from engorgement, erythema and œdema. It rarely happens that these circulatory disturbances in the skin of the lid form an isolated clinical picture sui generis, usually they are concomitant phenomena of other diseases, of which, as a rule, they indicate the presence. Therefore their diagnostic value must be briefly considered.

I. Hemorrhages in the Skin of the Lid

Hemorrhages in the skin of the lid, partly in the cutis, partly in the subcutaneous tissue, are called sugillations when extensive, petechiæ when divided into several spots. Sugillations are indicative of injuries, such as operations, penetrating wounds, or contusions. They may be due to fissures and fractures of the bones of the orbit, and very often are symptomatic of fractures of the skull. Frequently they are an evidence of fracture of the roof of the orbit. Extensive sugillations of the skin of the lid are also met with in grave cases of crushing of the thorax and abdomen. A backward engorgement of the venous blood, that causes rupture of the little veins, is produced by these compressions, which are not infrequently met with in railroad accidents. A trivial injury may produce an extensive hemorrhage in the lids in hæmophilia. Petechiæ usually indicate the hemorrhagic diathesis, scurvy, purpura hæmorrhagica, morbus maculosus, *Barlow's* disease, or sepsis.

II. Arterial Hyperaemias of the Skin of the Lid

Arterial hyperæmias of the skin of the lid are met with in grave inflammations of the conjunctiva, as in gonorrhœal conjunctivitis, or in this case of panophthalmitis, in which the skin of both eyelids is much swollen, glistening, tense, smooth, red, and hot to the touch.

This active hyperæmia accompanies serious intraocular suppuration, cellulitis of the orbit, and the infectious diseases of the conjunctiva. An indication of the seat of the disease may often be obtained from the situation and extent of such an inflammatory swelling.

If this swelling is confined to the medial side of the lower lid it indicates a cellulitis of the lacrymal sac; if it is situated above the ligamentum canthi internum of the upper lid, an empyema of the frontal sinus is suggested. Empyema of the antrum of *Highmore* and periostitis of the margin of the orbit may also be indicated by such local inflammatory swellings, which also form a symptom of considerable importance in one of the most common diseases of the lid, hordeolum.

III. Engorgement Hyperaemia of the Skin of the Lid

Engorgement hyperæmia of the skin of the lid furnishes quite a different diagnostic indication. When the venous circulation is obstructed, the veins become prominent, tortuous, dilated and proclaim the trouble, not rarely in conjunction with a swelling of the skin and a heaviness of the entire lid. The cause of such a venous stasis may be general or local.

Among the general causes may be mentioned the patent foramen ovale of the heart, and a stenosis of the pulmonary artery. The bluish red, puffy cyanosis met with in these cases is very characteristic.

When the venous stasis of the lids is confined to one side of the face the cause is to be sought among such local factors as pressure of the cervical glands upon the common jugular vein, thrombosis of the orbital veins, or the pressure of retrobulbar tumors upon the venæ palpebrales. In children a long continued blepharospasm frequently causes an œdematous swelling of the lids with venous stasis, because the veins passing through the orbicularis have been compressed.

In anæmia and chlorosis the edges of the eyelids are apt to be bluish and the vessels of the skin of the lid to be partially dilated. We also meet with a dilatation of both the arteries and veins of the lid in arteriovenous aneurysm of the internal carotid and the cavernous sinus, which causes pulsating exophthalmos.

The arteries alone may be greatly distended by aneurysms in the vicinity of the lid. A dilatation of the veins is not rarely indicative of a retrobulbar tumor, or of exophthalmic goitre.

Almost all of the vascular tumors of the lid consist of dilated veins. When such an angioma results in a saclike stretching of the lid we speak of a varicoblepharon.

IV. Erythema

This patient has developed a circumscribed erythema about the eye after wearing a moist dressing for a short time. The same condition may be produced by the application of hot poultices. Stokers and smiths develop slight erythemata on the skin of the lids under the influence of radiant heat. The erythema of the face produced by alcohol, tea, coffee and many febrile diseases is known to everyone.

We also meet with an urticaria on the skin of the lid. Persons so afflicted need only to rub the eye a little to cause the purple red, elevated spots to appear.

V. Oedema

Finally we return to œdema, whence we started. It is necessary only to mention the œdematous swellings of the lids from hydræmias in the course of cachexia, or nephritis, in which the skin of the lid takes the form of a sac, or cushion. In lymph stasis the lid feels soft, almost fluctuating. Lymphangiectases have also been observed on the lids.

70. Hordeolum Externum and the Commoner Acute Exudative Inflammations of the Skin of the Lid

This patient has an acute inflammation in his upper lid which is sufficiently painful to deprive him of sleep. Near the margin may be felt a point of resistance that is very tender. The skin over the prominence is red, hot and yellowish at its top, where the pus shines through. Every layman knows this as a stye; we call it a hordeolum.

I. Common Local Infectious Inflammations of the Skin of the Lid

The *hordeolum externum* is a staphylococcic infection of a sebaceous follicle, and is one of the forms of acne. The manner in which a purulent infection of a sebaceous follicle takes place is easily understood, as staphyloccocci and other microorganisms normally occupy the margin of the lid, the uppermost layers of the epithelium, the efferent ducts of the gland, and the cilia. Hordeolum externum is principally met with about the age of puberty, particularly when a chronic inflammation of the margins of the lids, a chronic conjunctivitis, anæmia, or chlorosis forms a predisposing cause. The cocci are inoculated into the gland by rubbing the lids, and there cause a localized, painful, inflammatory infiltrate in the skin, associated, in proportion to its extent, with an inflammatory swelling of the lid. Soon a pustule develops from this infiltrate, which may either dry up or rupture, after which the inflammation and swelling subside. *Acne varioliformis* and *necroticans* are met with more rarely; they start in a gland of the hair follicle. Acne necroticans may attack all four lids and may be distinguished from an ordinary hordeolum by a dry, necrotic scab which is firmly adherent to the subjacent tissue.

The treatment of hordeolum is to open the little abscess by a small incision and then to cover the eye with wet dressings for a few days. During an early stage the use of hot compresses may be indicated. The disease lasts only a few days, but is very annoying. When the virulence of the infection is very great the preauricular gland becomes swollen and the conjunctiva chemotic. Hordeolum externum is caused by suppuration of one of *Zeiss'* glands, hordeolum internum by the formation of an abscess in a *Meibomian* gland.

The *furuncle of the skin of the lid* differs from the preceding only in degree. It also is a staphylococcic infection and may start in either a sebaceous, or a sweat follicle, more often in the former. A little nodule is formed, having in its middle a lanugo hair, and from this soon develops a marked infiltration. When the furuncle finally ruptures, a craterlike opening is formed that contains the well known core of necrotic tissue. A furuncle that arises in a sweat gland and begins as a hard infiltration deep in the tissue is much more serious.

The carbuncle of the eyelid, which begins in the region of the brow, is directly dangerous to life. It consists of a conglomerate of furuncles and is dangerous because it may spread both superficially and deeply. In very favorable cases a deep abscess cavity is formed, after rupture, which heals with an extensive cicatrization that may result in an abnormal position of the lid. After rupture of the abscess the skin may be perforated like a sieve. A carbuncle may also cause thrombophlebitis of the orbital vein, purulent basal meningitis, or a pyogenous general infection. All these inflammations start from an eczematous disease of the lid and their malignancy is increased by disturbances of the metabolism, such as diabetes, or marasmus. When such a furuncular inflammation is of a serious nature the general condition is affected and in addition to the beating pains in the lid we meet with fever and swelling of the preauricular gland. The possibility of an infection with anthrax must be borne in mind in every case. Rapid and proper surgical treatment is indicated; a free incision, drainage and antiseptic dressings are necessary.

Another disease, the subcutaneous phlegmon, or *abscess of the lid*, demands particular mention because it is involved in other questions of differential diagnosis. The swelling, fluctuation and perforation of the pus enable us to recognize the clinical picture at once. Such an abscess of the lid may naturally come from an ectogenous infection, when the inflammatory agents enter through wounds in the skin, and we meet with such abscesses in people who suffer from panaritium, but an abscess of the lid may also be caused by an extension of a neighboring inflammation. It may come from a cellulitis of the orbit, or from a periostitis of its margin, or it may indicate that an empyema of the frontal sinus, or of the ethmoid, is seeking its exit into the orbit.

II. The More Frequent Haematogenous Dermatoses of the Skin of the Lid

In *varicella*, which attacks the skin of the lids by preference, little round, or acuminate vesicles are to be seen to rise from a reddened base. The contents of these vesicles are at first clear, but later become slightly turbid. The prognosis is good, the vesicles dry and form crusts in a few days. Usually the subjacent skin regains its normal appearance and we have only to try and prevent infection from scratching with the finger nails.

In measles the sharply defined roundish spots are to be found on the eyelids, which are swollen, in addition to the characteristic photophobia and conjunctivitis.

In scarlet fever the fine red spots with pale areolæ, which at first lie close together, soon blend into a uniform scarlet red and the epidermis peels from the lids after the redness has passed away.

Toxic dermatoses are also met with. Erythema exudativum occurs as one of the symptoms of pellagra, a disease that is widespread in Italy, Roumania and Bulgaria, and is considered to be a chronic intoxication induced by the ingestion of damaged maize. On the lids it produces an erythema with a formation of vesicles, scaling and pigmentation.

Occasionally we have to deal with *pemphigus*. It has been mentioned already that several diseases are included under this term. We distinguish clinically between an acute and a chronic form, in both of which large, clear bullæ are formed on the skin of the lid, with contents that gradually become turbid. The bullæ rupture and their coverings are cast off, leaving red, moist places. The surrounding skin is much reddened. Acute pemphigus may run a fatal course as pemphigus hæmorrhagicus; the chronic form is frequently associated with the same disease of the conjunctiva, or it may be preceded by the latter.

Urticaria rarely appears on the lids in the form of itching wheals, but more frequently as a marked œdema. It is met with very often. Various drugs, such as antipyrin, iodine, arsenic and chloral, as well as certain foods, like crabs and strawberries, may excite urticaria in persons subject to an idiosyncrasy. Cases of urticaria which have a reflex origin are also met with.

71. Vaccinola of the Margin of the Lid, and Some Rarer Infectious Inflammations

Both lids of one eye of this child are very œdematous and red. Near the outer canthus, in exactly corresponding places, on the margin of each lid, is a roundish, or oval, ulcer covered by a gray coating. The preauricular gland is inflamed and swollen. This is one stage of a vaccination pustule and these questions immediately arise: How comes a vaccination pustule to be met with on the margin of the lid? What is its course and of what serious importance is the disease to the eye?

I. Occurrence of Vaccinola

The child had been vaccinated, scratched his arm, then rubbed his eye and so transferred the infection. Vaccination takes very readily in the intermarginal space, evidently because the epithelial covering is very delicate, always moist and easily injured. Usually the infection is transmitted from vaccinated persons to others. Thus mothers and nurses infect themselves from vaccinated children. It is not uncommon for overfond mothers to lay their faces against the vaccinated arms of their children at the very time they are suffering from the effect of the vaccination. Even physicians have suffered from such pustules when they had carelessly brought the lymph into contact with their eyes.

II. Course of Vaccinola

A portion of the margin of the lid begins to swell and a papule appears at this point, which develops in a few days into a pustule, while the symptoms of inflammation increase. The pressure of the lids together soon causes the pustule to rupture, and from this point its course differs from that of a vaccine pustule elsewhere in the skin. It does not dry up, because it is kept wet constantly by the conjunctival fluid, but an ulcer is formed, as in this case, which is covered by a gray white coating. The eyelids in the meantime have become œdematous and hard, while the amount of the conjunctival secretion has become somewhat greater. Sometimes several pustules appear and in some cases the entire margin of the lid may be studded with such ulcers. Usually they disappear in from eight to fourteen days from this time.

III. The Importance of Vaccinola to the Eye

Fortunately the prognosis is good in most cases. The cilia fall out where the ulcers are formed and, although they may grow again, sometimes the loss is permanent. In many cases the cornea becomes involved, especially when a defect was present in the epithelium, and a keratitis develops which resembles to a certain extent keratitis disciformis. A delicate gray disc is formed, its margin denser than the central part, which is sometimes surrounded by parallel rings. At the same time symptoms of serous iritis appear, with deposits on *Descemet's* membrane. Permanent opacities that harm the vision are left only in very severe cases. The prognosis of vaccinola of the margin of the lid is generally good.

There is nothing to be done in the way of treatment except to cleanse the eye regularly and to apply ointments of boric acid, or of sublimate.

The consequences of *true small pox on the lids* and on the eye are very different, but thanks to the results of protective vaccination we no longer meet with this disease. In variola the characteristic papules develop likewise on the margin of the lid and become pustules, but the inflammation is much more severe and penetrating, and the result is marked cicatrization of the margin, or surface of the lid. The cilia are permanently lost; the entire margin of the lid may be broken down by the confluent ulcers; trichiasis, distichiasis and entropion may be the consequences. As the pocks may be situated on the scleral conjunctiva this disease is one of extreme danger to the eye. Even the ophthalmologist has every reason not to long for a return of the good old times when small pox raged unhindered throughout the land, and we shall always continue to exert the whole of our power to extend still more widely the protection afforded by vaccination, in spite of all the efforts of opponents of this beneficent measure.

A vaccination pustule on the margin of the lid might be mistaken for an ulcus molle, but the history will guard against this error. The soft chancre of the eye is an extremely rare disease which forms a fatty, yellowish, suppurating ulcer on the margin of the lid, or at the canthus, with very red, sharply excavated, but soft margins. The preauricular gland is painful and swollen.

A rather more common infection of the skin of the lids in certain callings is anthrax, which may appear in the form of either a diffuse anthrax œdema, or of an anthrax carbuncle, which has already been briefly mentioned. Anthrax œdema of the lids has a great tendency to spread over the skin of the face, head and neck. The color of the skin is at first pale, its consistency doughy; later the lid becomes very cyanotic, while bullæ appear and necroses of the skin are formed. The anthrax carbuncle starts as a little itching, or burning, red spot which develops into a pustule. A crater shaped ulcer is formed by a rapid destruction of tissue with severe symptoms of inflammation. Infection takes place through the rubbing of anthrax bacilli, or spores, into a little wound in the skin of the lid. This disease is met with in people who deal with animals, or with material that comes directly from animals; shepherds, butchers, tanners and veterinarians furnish the bulk of the cases of this very malignant disease. Anthrax causes a great destruction of the lids, which leads to extensive cicatrization. Its mortality is about 25%. The diagnosis can be rendered positive only by the demonstration of anthrax bacilli, for which cultures and inoculations of mice with the tissue are necessary. I mention the disease again at this place because it may be mistaken for a grave furuncle, or carbuncle of the lid. Treatment consists of anthrax serum, injected intravenously and in the neighborhood of the infection, absolute rest and antiseptic dressings.

Another infection of the skin of the lid, which is likewise to a certain degree an occupation disease, is glanders. This infection comes from the nasal mucus and pus of horses suffering from glanders, and its course may be acute, with tempestuous symptoms of inflammation, or chronic. In the former case an infiltrate resembling that of a carbuncle is associated with a great swelling of the skin of the lid and of the lymphatic glands in the vicinity. In chronic glanders large nodes and cordlike thickenings appear in the lid and give rise to sluggish ulcers. The prognosis is very serious. A disease of the lids caused by actinomycosis, or blastomycetæ, is very rare, and we also need not go into details here concerning the Oriental boil.

Finally, extensive *necroses of the skin of the lid* occur in quite a number of diseases, in the form of dry, or moist gangrene. In dry gangrene the dark skin of the lid is changed into a black, crusty mass; in moist gangrene the surface of the lid becomes ulcerated.

On one occasion I saw gangrene of the lid follow varicella in a little child. The skin of the upper lid became necrotic from the brow to its margin and from the outer to the inner canthus; the muscles and the tarsus were also partially destroyed. It always seems to be characteristic of gangrene of the lids that only the surface is affected and that the margins are spared, even when both lids are involved. Evidently the margin is the portion of the lid that is most richly supplied with blood vessels and is therefore the best nourished. Necrosis of the delicate skin of the lid may naturally be produced by various agencies. Take for example a case of blennorrhœa neonatorum which came under our observation recently. The child had been subjected to energetic treatment with applications of ice to the lids, which had caused the skin of the lid of one eye to necrose. I mention this case as a warning that ice should be used carefully in inflammations of the eye.

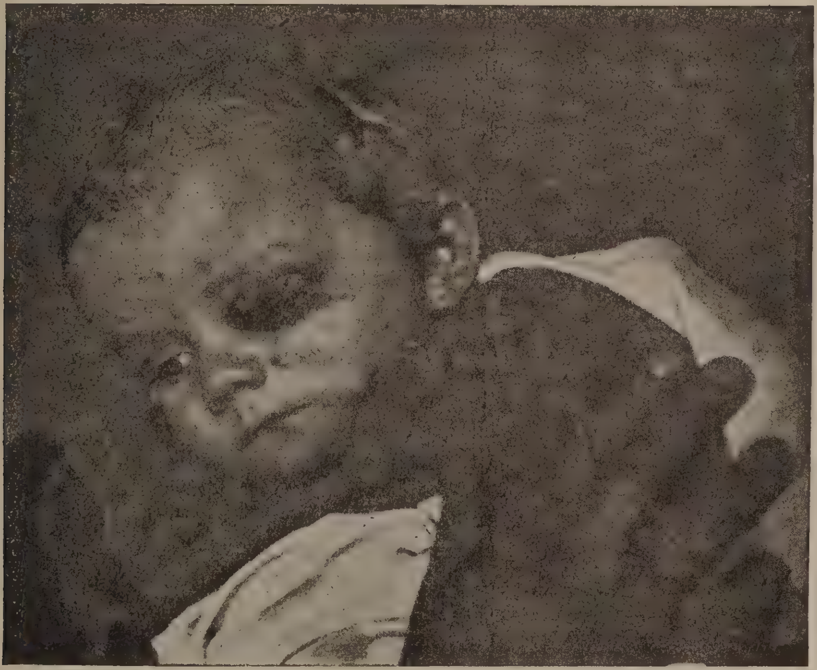

FIG. 55.—Gangrene of the Lid in Varicella.

Contusions and lacerations naturally predispose to necrosis. Necrosis is usually due to secondary infection by streptococci, staphylococci, diphtheria bacilli and germs of putrefaction, as in measles, scarlet fever, impetigo, eczema and varicella. Necroses of the lids have been observed in septicopyæmia from metastasis. Gangrenous stomatitis, noma, may also be transferred to the lids.

In addition to these infectious causes a general nutritive disturbance may produce necrosis of the lid in rare cases. Such necroses have been met with in alcoholism, diabetes, syphilis, and typhoid marasmus. The skin of the lid may also undergo a dry gangrene in the course of a grave herpes zoster. General treatment is the main reliance in all these forms of gangrene. The casting off of the sloughs must be awaited, while the eye is dressed with ointments, or antiseptics.

In addition to these more acute inflammations a great number of chronic morbid processes are met with which are chiefly of dermatological interest and so may be mentioned very briefly. 1. *Psoriasis vulgaris.* This is rarely met with on the eyelids. The round elevations surrounded by a delicate areola and covered with glistening scales, can be easily recognized. 2. In *pityriasis rubra* the skin of the lid is reddened and very scaly. As this disease may lead to an atrophy of the skin with contraction, its sequel, in the form of ectropion, may call for ophthalmic treatment. 3. As *lichen ruber planus* appears on the face very rarely, it is scarcely to be met with on the eyelids, but in extensive *lichen ruber cuminatus,* which may render the skin of the whole face uniformly thick and scaly, abnormal positions of the lids have been repeatedly observed. The same is true of *lupus erythematosus,* which ordinarily starts from the bridge of the nose and spreads to both sides of the face, and may readily cause ectropion by its cicatrization. Such rare diseases as rhinoscleroma, mycosis fungoides, frambœsia tropica and lepra may be mentioned, but they are of no interest to us here.

72. Seborrhœa of the Margin of the Lid

The most striking symptom in a case of this nature is the redness of the margins of the lids; the eyes seem to be edged with red. Although the margin of the lid is in fact only a part of the skin, we consider it separately because it contains the cilia, with their hairbulbs and glands, and because its diseases are among the most common with which we have to deal, so that they need to be described rather more accurately.

If we would diagnose correctly a disease of the margin of the lid we must consider it from the dermatological standpoint, and not be content with the names blepharitis squamosa and ulcerosa, which are frequently employed. I advise for both clinical and didactic reasons that this question be answered first, whenever dealing with a disease of the margin of the lid: Is this a disease of the cilia, or of the hairbulbs?

To answer this question we cleanse a portion, or the entire margin, of the lid and consider the condition of the skin. In conjunctivitis the secretion of the conjunctiva may dry on the margin of the lid, but, when this has been removed, the skin of the lid will appear to be normal, at least at the commencement of the disease. In the patient now being studied the skin between the cilia, and in their vicinity, is covered by gray and white scales that greatly resemble dandruff. If these scales are removed with a bit of wet cotton, or by rubbing with dry cotton, the margin of the lid is seen to be somewhat reddened, the skin intact, though hyperæmic, and full of minute blood vessels. The conjunctiva tarsi is also reddened. The cilia are intact and no pus or ulceration is present; thus a disease of the cilia is excluded and the seat of the disturbance is to be sought in the sebaceous follicles.

The condition is one in which the function of the sebaceous follicles is increased. We call it seborrhœa of the margin of the lid.

The form is sometimes that of seborrhœa oleosa, in which the cilia, eyebrows and frequently the scalp, appear as if they had been anointed with fat; more commonly it is that of seborrhœa sicca, characterized by the formation of whitish, branlike scales. This disease is extremely widespread and is met with in many people, especially blondes.

Its origin has not yet been satisfactorily explained. Some dermatologists think it is due to infection and a sebotactic influence has been ascribed to certain bacteria. It has frequently been supposed that a connection exists between seborrhœa and errors of refraction; uncorrected hypermetropia may increase the redness of the margins of the lids and so favor seborrhœa, but although we admit that the abnormal innervations exerted by the errors of refraction may influence the vascular supply, yet, as *v. Michel* has insisted, there need exist no causal connection between seborrhœa of the sebaceous follicles and the refraction. Many things favor the supposition that seborrhœa sicca is a parasitic disease.

The general constitution is of great importance. Anæmia and scrofulosis are not rarely met with in children who suffer from this disease, and it can scarcely be accidental that it is met with so often in persons with delicate skins and light, or red hair. Every adult knows that bad air, or a wakeful night increases the redness of the margins of the lids.

Seborrhœa follows an extremely chronic course and usually persists in a slight degree throughout life. Yet it can be ameliorated and often cured for a long time by proper treatment. It should be treated by the general practitioner. Such general conditions as anæmia and scrofulosis should be corrected first; then the patient should be instructed how important it is to cleanse the margins of the lids night and morning. Before retiring for the night an ointment should be applied to the cleansed and dried margins, but it is not every ointment that will be borne by such eyes. Yellow ointment is frequently irritating; sometimes the simple boric acid ointment is sufficient. Another one which may be prescribed is composed of: Lac sulphur, 1.0, or resorcinum albissimum, 0.5; zinci oxidum, amylum purum, āā, 10.0; vaselinum, 20.0. It goes without saying that any existing chronic conjunctival catarrh must be treated. When the scales are adherent we may use a sharp spoon very carefully for the first thorough cleaning.

73. The So Called Blepharitis Ulcerosa and the Diseases of the Cilia

The first thing to be determined, when we examine a case of disease of the margin of the lid, is whether the disease is one of the cilia. There is no doubt that the cilia are diseased in the three cases I show you today.

Case 1.—In addition to the redness and the dry crusts the entire margin of the lid is covered by round ulcers, or little abscesses from the middle of each of which a cilium protrudes. In some places these ulcers have become confluent, so that a large part of the margin is transformed into a raw surface.

Case 2.—This child has scrofulosis and is suffering from keratoconjunctivitis, in conjunction with an extensive eczema of the face that involves the skin and margin of the lid.

Case 3.—This third patient, an old woman, says she has suffered from sore eyes for more than ten years. In her we see the consequences of a chronic catarrhal conjunctivitis with stenosis of the lacrymal duct. The intermarginal space is no longer present, part of the cilia are lost, the remainder are directed toward the eyeball, are partially stunted, and scrape the opaque cornea with each movement of the lid. Hitherto the more or less satisfactory designation of such cases as these has been blepharitis ulcerosa; but this term has been made to include quite a number of very different diseases.

A. Normal Behavior of the Cilia

The cilia, or eyelashes, are implanted in the outer edge of a normal lid margin so that those on the upper lid are directed concavely upward, those on the lower lid concavely downward. Their anatomical construction is that of stiff hairs, and we designate the portion protruding from the skin the trunk, that within the skin the root of the cilium.

Microscopically the trunk consists of the nonmedullated fibres of the cuticle, the fibre cells, and internally of medullated fibres, the medulla cells. Pigment granules and dissolved pigment are found in the cells of the cortex. The root is surrounded by a hair follicle and sheath of connective tissue. Sebaceous and sweat glands open into the ciliary follicle. The root begins with the swelling of the hairbulb over the hair papilla, which presents a connective tissue cone. We differentiate also an outer and an inner root sheath.

It has been calculated that under normal conditions a cilium is cast off and replaced by a new one in about 150 days. A slight rubbing of the lid is often sufficient to cause loose, old cilia to fall out. As the root of an old cilium is club shaped these are spoken of as club hairs to distinguish them from the papillary hairs of the scalp.

B. Diseases of the Cilia

When we consider diseases of the cilia in detail we find that the following lesions may be present: 1, a change in the structure of the cilium; 2, mycosis; 3, a follicular or perifollicular inflammation of the hair follicle; 4, anomalies in the growth and direction of the cilia. The third and fourth groups are often combined.

I. *Structural Changes of the Cilia*

Certain changes in the structure of the cilia are very striking. They may be congenitally white. We know that the color of the cilia, like that of the hair, depends on the pigment and air they contain and on the condition of the surface. In congenital whiteness of the cilia, called albinism, the hair cells are devoid of pigment from their roots. Only a portion of the cilia may be affected by such a *leucosis*. The cilia acquire a light color in many ways. They remain dark longer than the hair in the bleaching due to age, but finally become more or less gray. In premature age, which is hereditary in many families, or may result from sickness, they may become gray earlier. A phenomenon that has always been of special interest is that the cilia may become white very rapidly, so to say over night. Cases have been reported in which the cilia became white suddenly as the result of a fright. It may be imagined that the effect of the nervous and circulatory disturbance is to deprive the hair papilla of its power to form coloring matter and to cut off the supply of pigment to the hair trunk.

Cilia have also been observed to become white in the course of sympathetic iridocyclitis, after operations on the eye and in trigeminal neuralgia, and many cases of spontaneous poliosis have been met with for which no cause could be assigned. Such people often dye their lashes.

Cilia are frequently dyed that have lost their color. Many nations are accustomed to dye their lashes and eyebrows and I need not mention that this bad habit is not unknown among ourselves. In many branches of industry the nature of the occupation is such as to cause the eyelashes to become stained.

The cilia may undergo other structural changes. The root may be softened by inflammatory disease of the hair follicle and occasionally a nodular formation, trichorexis nodosa, appears on individual lashes and forms punctate nodes on the hair trunk.

II. *Mycosis of the Cilia*

The mycoses met with in the cilia are sycosis parasitaria and favus.

Sycosis parasitaria is caused by a species of trichophyton. The fungus enters the hair follicle, proliferates with its gonidia between the cuticle and the cortical layer, and excites a severe inflammation which finally results in a perifollicular abscess. Pustules with large inflammatory nodes are formed at the bases of the lashes, which protrude from the little abscesses. When the crusts are removed the pus is evacuated. A differentiation must be made between sycosis parasitaria and coccogenes, of which I shall speak later. When the presence of sycosis parasitaria is suspected we have to demonstrate the fungus on the epilated cilia. The treatment is the same as for sycosis on other parts of the body.

Favus is extremely rare. A dry, dimpled disk is formed in the skin by the proliferation of the hyphomycetes (achorion *Schœnleinii*). Beneath the scab the skin is peculiarly moist and lustrous. Treatment consists of the removal of the scab and the application of sublimate or resorcin ointment.

Parasites are also met with on the margin of the lid. In many cases an irritation of the margin is due to the pediculus pubis, the black nits of which may render the margin so dark as to attract the attention of the physician. These parasites are easily removed by inunctions of unguentum cinereum.

The acarus folliculorum, or demodex, may be found on the roots of the lashes in many men with perfectly healthy eyes. This parasite is usually harmless and excites an irritation of the margin of the lid only in rare cases, when the use of a weak ointment of balsam of Peru is to be recommended.

III. *The Perifollicular and Follicular Inflammations of the Hair Follicle*

The overwhelmingly great majority of cases of so called blepharitis ulcerosa, are perifollicular and follicular inflammations of the hair follicles, and are the consequences of either an eczema of the margin of the lid, or of a sycosis produced by staphylococci.

(*a*) *Eczema of the Margin of the Lid*

We cannot identify eczema of the eye by itself with eczema of the skin. Yet keratoconjunctivitis ordinarily starts in the course of an eczematous disease of the skin, particularly of the face and lids, and the physician can prevent, or ameliorate this disease of the eye by timely and proper treatment of the eczema of the lid.

The theories concerning eczema still conflict. We cannot go in detail into its etiology, but will merely note clinically the following facts:

A general cause and local factors participate in the origin of eczema.

There is certainly a special condition and predisposition of the skin, to which are to be added nutritive disturbances, anæmia and scrofulosis. Among local causes may be included all possible irritations, and not a few believe that it has a parasitic origin.

Eczema of the lids may either pass through all the characteristic forms and stages of the disease, or a certain form of the clinical picture may predominate throughout, just as elsewhere on the skin. It may begin with either a diffuse redness of the skin, or with numerous little nodules, which correspond to the mouths of the follicles from which little vesicles may develop. The contents of these vesicles may either dry up and scale off, or become turbid and purulent; in the latter case the disease is known as eczema pustulosum. If the pustules become confluent and rupture, large erosions are formed from which serous fluid is constantly secreted. These places are very red; eczema rubrum or madidans. If the fluid coagulates, thick crusts are formed and we have eczema crustosum, or impetiginosum. As the erosions heal they become covered with epithelium, which for a long time may be desquamated in the form of whitish scales: eczema squamosum. The first thing to decide in our case is what form of eczema is present.

The situation of the disease varies greatly. It may spread from the neighboring skin to that of the lid, or it may be confined to the latter. The skin of the lid is diffusely red and swollen, in obstinate cases and recurrences it may be extremely thickened so as to lead us to speak of a pachydermatic eczema, or an eczema callosum of the lids. Analogous conditions of thickening are met with in the skin of the lips and nose. The usual situation is at the canthus, where the wetting of the skin may readily give rise to a moist eczema. It is hardly necessary to recall to memory the importance of rhagades. Such an eczema is also called eczema intertriginosum and is found on the skin of the lid where the covering fold overhangs the rest.

In our patient the margins of the lids are red and swollen, the cilia are glued together by the dry secretion and the entire margin is covered by thick crusts. Frequently pustules are formed, each with a cilium in its centre, and then the clinical picture of a sycosis is more or less nearly approached. We imagine that the eczematous places very easily become the seat of secondary infections, the liability to which is greatly increased by rubbing the lids, for the itching and other annoying sensations naturally cause such patients to wipe and rub their eyes. The disease is introduced through the constant wetting of the skin and the maceration of the epidermis.

An eczema of the margin of the lid which has existed for a long time results not only in a rapid change of cilia, but also in the serious lesions in their bed which are exemplified so dreadfully by our third case. It is therefore very important that a physician should be skilled in the treatment of eczema of the lids.

The patient must first be taught not to struggle against the itching. In order to prevent children from scrubbing their eyes we place short pasteboard splints about their elbows. This looks more cruel than it is, for the children can still play.

In the second place care must be taken that the skin of the lid, whether at the canthus or on the margin, is not macerated by the secreted conjunctival fluid, because otherwise the eczema will continue to spread. For this purpose the skin and margin of the lids are anointed at regular intervals with pure vaseline immediately after they have been dried. It goes without saying that the conjunctival troubles which occasion an abnormal secretion must be treated at the same time. Many cases will not bear even boric acid ointment. When these measures have been taken everything possible has been done to prevent the outbreak, or the extension of an eczema.

If the question is how to do away with an existing eczema, the scabs and scales are soaked off, or loosened by an application of grease, and removed so as to cleanse the lid. Then we are guided by the form of the eczema; thus in erosions, or eczema madidans, an excellent effect is obtained by painting the surrounding skin daily with a 1% solution of silver nitrate and then smearing the lids with vaseline or boric acid ointment. The applications of powders have also been recommended.

In all forms subsequent treatment with pastes is indicated. Primarily we order here *Lassar's* paste: acidum salicylicum, 0.3; zinci oxidum, amylum, āā, 3.0; vaselinum album ad., 10.0. Eczema is very amenable to treatment, and it is well to remember, as soon as a so called blepharitis ulcerosa is seen, that an eczema is the primary cause of the disease in very many cases.

(*b*) *Sycosis Staphylogenes or Coccogenes*

In this other case we find on the margin of the lids pustules, each perforated by a cilium. After the pustules have broken roundish ulcers are left, each with a cilium protruding. If such a cilium is pulled out it is found to be loose, with its root sheath swollen and looking purulent. At first the pustules are single and may occupy for a long time only a portion of the margin of the lid, but finally they spread over its entire surface. Such a sycosis may originate independently of an eczema of the skin. If we remove the scabs and dry masses of pus we have before us a raw surface covered with ulcers.

This disease is produced by staphylococci that have been rubbed into the open mouths of the follicles. The stages of such an infection are, impetigo of the funnel of the hair follicle, perifollicular infiltration followed by abscess, and suppuration of the hair follicle.

An analogous picture may be seen on the margin of the lid in pustular eczema, but in such a case other symptoms of eczema are to be found in the vicinity. In this patient the rest of the skin of the lid is perfectly healthy. In order to make the diagnosis positive the contents of the pustule and the diseased cilia are examined in search of a sycosis parasitaria.

According to my experience the treatment of all these cases of perifollicular and follicular inflammation of the margin of the lid should be mild. Application of the stick of silver nitrate, which is frequently recommended, is proper only in obstinate and malignant cases. We simply open the pustules, epilate the cilia in the most diseased parts, and smear the margin regularly with boric acid ointment. Tannin and sulphur ointment has also been recommended.

IV. *Disturbances in the Growth of the Cilia*

Such an inflammation of the margin of the lid has evidently existed a long time in our next case; it has penetrated deeply and induced serious nutritive disturbances of the cilia. The intermarginal space has been almost wiped out, the outer and inner edges of the lid are almost blended. Three symptoms attract our attention when such a great change has taken place in the bed of the cilia.

1. We may find signs of hypertrophy of the cilia, and then we speak of hypertrichosis. Single very long lashes are occasionally met with, or the number of cilia may be abnormally great. In rare cases we meet with a con-

genital polytrichia, a condition in which perfectly normal lashes are arranged in several rows. It is probably due to a congenital abnormality of development through which cilia were developed in the place of the *Meibomian* glands. In acquired polytrichia there is an irregular and confused growth of cilia here and there, where they do not belong, which have come from rudimentary, or pathologically altered hair germs. Such a polytrichia may appear in old age associated with an increase of hair, as in women; but such conditions are met with chiefly in cicatrization of the margin of the lid caused by trachoma.

2. More often we meet with a hypotrichosis, or alopecia. This condition also may be congenital, when it is usually associated with a universal alopecia and is due to an inhibition of development, but it may be acquired from both general and local causes. According to *Willbrand*, the falling out of the cilia is not rare in syphilis, and *v. Michel* saw a universal alopecia with absence of cilia and supercilia after an attack of scarlet fever. The lashes have also been observed to fall out in chronic arsenic poisoning, cachexia strumipriva and exophthalmic goitre.

Such a falling out of the lashes is met with in all diseases of the margins of the lids, seborrhœa, mycoses, follicular inflammations and cicatrizations, and, if one desires, a differentiation may be made between a seborrhœic, a mycotic, an inflammatory and a cicatricial alopecia. If the hair papillæ are destroyed, the loss of the lashes is permanent. It should be mentioned in this connection that hysterical patients not rarely pull out their lashes.

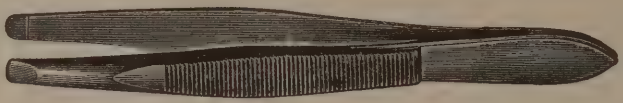

FIG. 56.—Cilia Forceps.

3. Oblique position of the cilia. Those disturbances in the bed of the cilia, which alter their direction, are of the greatest practical interest, because they soonest cause injury to the eye.

A cilium, or a bunch of cilia, may be turned outward over the edge of the lid and pressed into the skin of the intermarginal space. Such lashes are called cilia incarcerata; they are themselves normal, have been encapsuled by epithelial, or granulation tissue and may have their proper direction restored by an incision. The point of the lash may be sunk obliquely into the skin of the upper lid; this is called a cilium inversum. These conditions play a very subordinate part in the erroneous direction of growth which we call trichiasis, a very common trouble.

We understand by trichiasis an alteration in the bed of the cilia by means of which the lashes become directed backward and inward, downward on the upper lid, upward on the lower. It is often associated with polytrichia and alopecia.

The consequences may be seen in this patient. The badly directed lashes scrub the cornea with each motion of the lid. Their mildest effect is to irritate the eye and produce photophobia, blepharospasm and increased lacrymation. They may also cause defects in the epithelium of the cornea, which in turn are very painful, easily accessible to purulent infection and frequently result in ulcers. In other cases the constant mechanical irritation excites a proliferation of the epithelium and so starts a pannus.

I lay great stress on these conditions and demonstrate them carefully because the commencement of a trichiasis is so easily and so often overlooked. It is not always easy to see single, minute, misdirected cilia with the unaided eye, but they will not escape an examination of the margin of the lid with a loupe. If you will examine this patient with *Zeiss'* corneal microscope you will understand why such a margin of the lid has been compared appositely with an untended forest. Normally there is a layer of fluid where the inner edge of the lid touches the eyeball, which gives a reflex parallel to the margin; this uniform reflex is broken wherever a cilium is directed inward. All this is the result of blepharitis.

Patients with partial trichiasis may often be helped by regular epilation of the offending lashes, but the benefit can only be temporary, because the lashes return sooner or later. It is much better to destroy the hairbulb by electrolysis, when it is necessary to remove single lashes. A fine needle attached to the negative pole is introduced into the hairbulb, the positive pole is placed on the temple. The effect is shown by bubbles of gas rising along the root of the lash which can then be easily lifted from its bed; its growth is then destroyed.

When the trichiasis is of considerable extent, or involves the entire margin of the lid it can be obviated only by means of an operation. As it is often associated with an entropion the methods employed are usually those that will at the same time relieve the latter.

74. Blepharophimosis and Lagophthalmos, and Their Operative Treatment

Sometimes we have to enlarge the palpebral fissure when it is abnormally small and, inversely, to diminish it when abnormally large. A stenosis of the palpebral fissure, such as is presented in our first patient, is known as a blepharophimosis.

A. Blepharophimosis

It must be determined first whether the palpebral fissure is really contracted, for an apparent stenosis is not infrequently present. A vertical fold of skin may cover the outer canthus and a part of the palpebral fissure, but when it is drawn back so as to uncover the canthus, the fissure proves to be of normal size. Such a vertical fold of skin may become eczematous in a chronic conjunctival catarrh, or an irritated condition of the eye, from the constant wetting with the lacrymal fluid, and, as a result of this dermatitis, become hard and contracted horizontally by a blepharospasm, but after the conjunctivitis is well the dermatitis passes away and the apparent blepharophimosis disappears. In old people the skin may be relaxed and cover the outer canthus constantly. Our patient has a true blepharophimosis and we have to consider what causes operate to produce this condition.

Causes of Stenosis of the Palpebral Fissure

The palpebral fissure may be very small congenitally. Such a condition is met with chiefly in association with bilateral congenital ptosis, when the flat configuration of the face, the flat bridge of the nose, and the defective intelligence suggest the etiology.

It may be caused also by a congenital adhesion between the upper and lower lids, called congenital ankyloblepharon, which may be a purely epithelial adhesion, or connective tissue bands may extend from the margin of one lid to that of the other, when it is called ankyloblepharon filiforme. This abnormality is unilateral, as a rule, and is readily corrected by an operation. In all such cases the etiology can be determined from the history.

Acquired stenosis may be due to faulty innervation of the muscles. Paralysis of the levator or spasm of the orbicularis may cause a complete closure of the palpebral fissure, so it is necessary to ascertain whether the cause is of this nature.

It may also be due to a change in the form of the eyeball; the lids fall together in phthisis bulbi following injuries or intraocular inflammations. After enucleation of the eye in children the orbit may be backward in its growth and the palpebral fissure be very small.

The most common cause is a contraction of the conjunctiva, or of the margins of the lids. Cicatricial contraction of the conjunctiva from trachoma, pemphigus, and burns in the vicinity of the outer canthus, may produce a blepharophimosis. The lids may grow together when opposite places on their margins are made raw by ulcers, burns, or such diseases as lupus and diphtheria. In severe burns the margins of the lids may become adherent throughout their entire extent, producing the condition known as acquired cryptophthalmos. In such cicatricial cases adhesions exist, as a rule, between the conjunctiva bulbi and conjunctiva tarsi, and the ankyloblepharon is associated with symblepharon.

In our case the chronic conjunctivitis, that has lasted for many years, together with the inflammation of the margin of the lid, has produced the blepharophimosis.

B. Lagophthalmos

Enlargements of the palpebral fissure are grouped together under the term lagophthalmos. A symptom common to most of these cases is that the closure of the lids is more or less imperfect, so that the anterior surface of the eyeball is not properly covered and moistened. The causes are various; rarely it is congenital.

Acquired lagophthalmos may be caused:

1. By spasm of the levator, or, more commonly, by paralysis of the orbicularis.
2. By the position of the eyeball in the orbit. In exophthalmos from any cause the eyeball presses the lids apart.
3. By a tumor from the eyeball which protrudes through the palpebral fissure.
4. By enlargement of the eyeball itself, as in congenital buphthalmos, when the lids are no longer able to cover the enlarged anterior surface of the eye. In high myopia also the eye may protrude as if it were exophthalmic.

If the cause of the lagophthalmos is a disturbance of innervation, this is to be treated primarily. In facial paresis the eyeball should be protected at night by a dressing. Operative intervention is often necessary in this condition. In this case of exophthalmic goitre a tarsorrhaphy is necessary, because we know of no other method by means of which to obviate the exophthalmos. Hence for the protection of the eye we shall enlarge the palpebral fissure in our first case and shorten it in our second.

Canthoplasty

We draw the lids well apart and somewhat toward the nose, so as to bring the outer commissure of the lids well into view. The blunt blade of a pair of strong, straight scissors is introduced horizontally beneath the commissure, which is then divided by closing the scissors. When the lids are separated the wound is seen to be in the form of a rhombus, the lateral sides of which are formed of conjunctiva. Everything now depends on the way in which the mucous membrane is sutured through the skin. We seize the conjunctiva at the junction of the two branches of the wound, draw it outward, and attach it to the skin at the angle made by the incision. Other sutures are then inserted above and below to unite the skin and conjunctiva. If the conjunctiva does not heal into the wound in the skin the latter will quickly reunite. The main purpose of this operation is to divide horizontally the outer muscle of the lid.

This simple operation is employed not only when it is necessary to enlarge the palpebral fissure temporarily in an obstinate blepharospasm, but also to weaken the spasmodic contractions of the orbicularis by section of its fibres, or to relieve the pressure of the greatly swollen lids upon the eyeball, as in grave blennorrhœa of adults. It may also happen that the palpebral fissure is too small for an enucleation, or for the removal of an orbital tumor, when this horizontal incision at the canthus may be of assistance.[1]

Tarsorrhaphy

In tarsorrhaphy the palpebral fissure is shortened by uniting the margins of the lids. The operation of *Fuchs* is a great improvement on that of *v. Graefe* and *v. Walther*, who freshened the margins of the lid, at the outer canthus, by excising the bed of the cilia for the desired distance, and then sutured the lids together. We first introduce a metal plate into the conjunctival sac to support the lids while the incisions are made, and then split the lower lid into two laminæ, for the distance desired, by an intermarginal incision. From the inner end of this section a second incision is made vertically through the skin, thus forming from the outer portion of the lower lid a flap, the upper and inner borders of which are free. The cilia on the upper border of this flap are in our way, so we abscise them and their bulbs with scissors applied on the flat. Now we turn to the upper lid and make an intermarginal incision at the outer canthus. The bed of the cilia is then ablated by vertical cuts with the scissors at each end of the intermarginal incision, connected by another incision through the skin running parallel to the margin of the lid. Thus we have produced a raw surface on the upper lid to which it is intended that the skin flap of the lower lid shall adhere by its raw surface. Both ends of a thread armed with a needle at each end are then passed through the upper lid from the conjunctiva forward near its free margin, thus leaving the loop of thread to lie on the conjunctiva. The ends of the thread are now passed from the raw surface through the skin flap of the lower lid and tied over a glass bead. In this way the base of the flap on the lower lid is kept pressed against the raw surface of the tarsus of the upper one. A few more sutures are then introduced to unite the edge of the skin flap with the edge of the wound in the upper lid. When a tarsorrhaphy is necessary at the inner canthus we excise with forceps and scissors a strip of skin from both the upper and lower lid in such a way that the wounds converge toward the inner canthus and meet there in an acute angle. The margins of the wound are then united. In order to obtain a good result from a tarsorrhaphy both eyes must be kept quiet under a bandage for several days.

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[1] A valuable modification of this operation, suggested by *Agnew* and called by him *cantholysis*, is to make the canthotomy in the manner described, to then introduce the points of a small, strong, fairly blunt pointed pair of scissors vertically upward in the wound, between the skin and conjunctiva of the upper lid, so as to enclose between them the upper canthal ligament, which is then divided. To do this the upper lid is put on the stretch by being pulled toward the nose. The lid is relaxed very much more by this operation than by the simple canthotomy, and is particularly indicated when relief from the pressure of the lid upon the eyeball is desired.—*F.*

In ectropion the lower lid may be lifted somewhat by means of a tarsorrhaphy, but such an operation is frequently insufficient. The operation is often of use in lagophthalmos due to facial paresis.

75. Ectropion and Its Treatment

In both of these cases the lower lid is everted so that the conjunctiva tarsi is visible, though to different degrees and from different causes.

The first patient is an old man who has developed this malposition of the lower lid as the result of the chronic catarrh from which he is suffering. In the other patient the condition is due to a severe burn of the face. From these two cases we may study the etiology of ectropion and two different operations for its correction.

The first thing to be noticed is that the ectropion is of different degree in the two cases.

At first the inner edge of the lid stands out somewhat from the eyeball, a condition we call eversion of the margin of the lid. Then the margin of the lid gradually draws away from the eye and the ectropion becomes partial, or total. It may, for example, be confined to the outer portion of the lid, and may affect either one eye or booth.

From the moment the conjunctiva tarsi is no longer in apposition with the eyeball, but is exposed to the air, injuries are produced which render the condition worse. The conjunctiva tarsi does not bear well exposure to the air, but becomes hyperæmic, swollen and inflamed. The thickened conjunctiva presses the lid still farther from the eyeball and increases the ectropion.

The punctum lacrimalium is naturally turned outward from the eyeball with the margin of the lid, and epiphora is produced. The overflowing tears cause excoriations of the skin and a dermatitis, which renders the skin of the lid heavier and helps to pull it down. These factors play a more or less important part in all forms of ectropion, but in other respects the primary causes may be very different and they must be diagnosed if treatment is to be properly directed at the commencement of this disease.

There are four different kinds of ectropion:

1. Ectropium spasticum.
2. Ectropium paralyticum.
3. Ectropium senile.
4. Ectropium cicatriceum.

1. Ectropium Spasticum

Spastic ectropion is met with principally in children suffering from blepharospasm caused by an eczematous conjunctivitis.

In childhood the skin of the lid is still tense and elastic, and tends to draw the margin of the lid toward the margin of the orbit. If a marked swelling of the conjunctiva presses the lid outward from the eyeball the lid may be everted by a considerable spasm of the orbicularis. A complete eversion of the lid is not uncommonly seen when we separate the lids of such a child. Usually the upper lid is affected alone, less commonly both lids, or the lower one only. Once the lid is everted, the peripheral bundles of the palpebral portion of the orbicularis contract about the everted part of the tarsus and hold the lid in this position. A venous engorgement is produced and then the everted lid forms a thick, red swelling. Formerly such an ectropion with much swelling of the conjunctiva was called ectropium sarcomatosum, a designation that is superfluous and may give rise to error. The palpebral fissure is completely closed and all muscular efforts, in crying or squeezing, increase the ectropion.

In the beginning of a spastic ectropion the physician may first try to replace the everted lid by lifting up a vertical fold of skin, drawing down the margin of the lid and pressing back the swollen conjunctiva; then fixing it in place by means of a bandage. This conservative procedure is aided by treatment of the conjunctivitis, and the rendering anæmic of the conjunctiva by means of one of the preparations from the suprarenal capsules. A provisory canthoplasty is frequently needed.

Snellen's suture may be used in obstinate cases of ectropion of the lower lid. One end of a thread, armed with a needle at each end, is introduced at the top of the everted transition fold, passed behind the lower margin of the tarsus and then through the lid in front of the lower margin of the orbit out upon the cheek; the other needle is then introduced and passed in the same manner a few millimetres distant from the first. A second suture may also be placed near the inner canthus. The ends of the threads are then tied over a roll of cotton tightly enough to produce a slight degree of entropion. The eye is bandaged and the sutures allowed to remain for several days.

II. Ectropium Paralyticum

This form is met with in cases of facial paresis, and naturally the lower lid alone is affected. Treatment is directed primarily against the facial paralysis, while the eye is bandaged during the night to prevent desiccation of the cornea. If the facial paralysis does not improve, a tarsorrhaphy may ameliorate the condition of lagophthalmos, and *Szymanowski's* operation for ectropion may be recommended if it is necessary to elevate the lid still more.

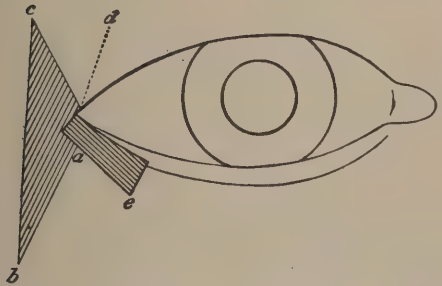


FIG. 57.—Operation for Ectropion after *Szymanowski.*

An intermarginal incision is made in the lower lid for a distance determined by the amount of shortening required at the outer canthus. A triangular flap of skin is then excised close to the outer canthus, as shown in the illustration. The skin near the margin of the wound (a b) is undermined, the points (a) and (c) are united and the raw surfaces covered.

III. Ectropium Senile

Several factors take part in the production of senile ectropion, such as is present in this old man. In the first place the strength of the orbicularis not infrequently fails, in such old people, until it can no longer hold the lower lid in apposition with the eyeball. In the second place the skin of the lids in such people is relaxed. In the third place a chronic conjunctivitis, a lacrymal disease, or a combination of the two may be added. In consequence of the weakness of the orbicularis, the weight of the relaxed skin and the swelling of the inflamed conjunctiva, the margin of the lid draws somewhat away from the eye, the eye lacrymates and the patient begins to wipe it, but instead of wiping it from below upward, to press the lid into place, he wipes it from above downward and increases its malposition. The extent of the tarsal conjunctiva that comes in contact with the air constantly increases and becomes thickened by papillary hypertrophy. The overflowing tears finally excite a dermatitis which by increasing the weight of the skin increases the ectropion. This is the condition in our patient. The hypertrophic conjunctiva is much thickened and contains numerous dark red, papillary elevations. If the condition is allowed to persist the conjunctiva gradually loses its character of mucous membrane, its upper surface becomes dry and lustreless, crusts form upon it and the epithelium appears almost horny.

At the beginning of this disease the patient must take care to wipe his eye always in the right direction. The chronic conjunctivitis and the lacrymal disease must be treated if present. When the conjunctiva tarsi is considerably thickened the application of the mitigated stick of silver nitrate forms an efficient remedy for an incipient ectropion. It should be applied, the casting off of the crusts awaited and then applied again. The use of the mitigated stick of silver in conjunctival diseases is highly objectionable, as a rule, but in this condition it often gives excellent results. An operation is necessary if an ectropion is too far advanced for this procedure to be of service.

The simplest operation is to excise a fold of the tumid conjunctiva. The incisions should converge downward and be carefully sutured. This is sufficient in many cases, but in others a more complicated method is required. *Kuhnt's* operation, as modified by *Dimmer*, is a very good one.

After cocain and adrenalin have been injected the middle of the lower lid is split along the intermarginal space. *Kuhnt* now excises a triangular piece of tarsus and conjunctiva from the ends of this intermarginal incision, its apex lying in the transition fold. If this wound is now closed with sutures a vertical fold will be formed in the skin. To avoid this and at the same time to shorten the lid more satisfactorily, the intermarginal incision may be continued to the outer canthus, a triangular piece excised from the tarsus and conjunctiva, and then a triangle of skin, with its base forming a prolongation of the palpebral fissure, may be removed near the outer canthus. The superabundant skin in the middle of the lid can then be slid toward the temporal side to fill in the defect. Thus a triangle of tarsus and conjunctiva is excised from the middle of the lid and a second triangle of skin from near the canthus.

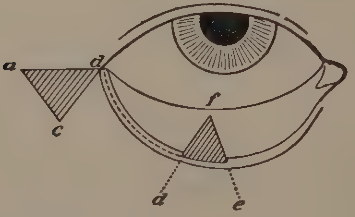


FIG. 58.—The Incisions.

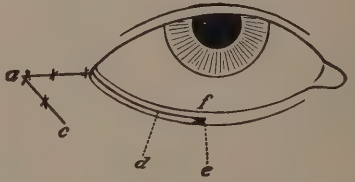


FIG. 59.—After Closure of the Margins of the Wound.

Operations after *Kuhnt* and *Dieffenbach.*

IV. Ectropium Cicatriceum

In this case the ectropion is due to the tension of the cicatricial skin of the lid. The degree and extent of a cicatricial ectropion varies greatly, as might be expected from the many causes that produce it. A considerable degree of tension induced by an eczematous inflammation of the skin may cause a slight degree of ectropion, but the diseases of the lids that are principally to be taken into account are those which result in necrosis and loss of tissue in the skin of the lids and face, such as wounds, burns and lupus. The cicatricial contraction of the skin of the face in lupus produces the highest degree of cicatricial ectropion. Anthrax, syphilis and gangrene of the skin of the lid occasionally produce it. It may also result from diseases of the bones at the margin of the orbit. It is not at all rare to see a fistula cause an inclusion of the skin in the bony cicatrix which finally leads to an eversion of the lid. Such bone disease and partial ectropion is usually met with at the outer canthus. It is evident from this that the treatment of a cicatricial ectropion must vary in different cases. If it is necessary to cover a large area after the cicatricial tissue has been extirpated we have at our disposal two procedures: blepharoplasty with pedicled or with nonpedicled flaps.

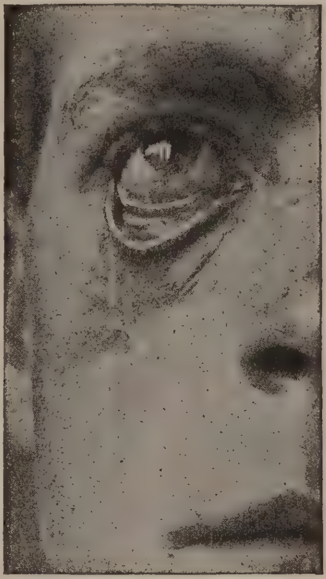


FIG. 60.—Cicatricial Ectropion.

Blepharoplasty with Pedicled Flaps

The advantages of this operation over that with flaps without pedicles are that the healing is more certain and that the contraction of the flap is not as great, but it has the disadvantage that the flap must be taken from the immediate vicinity of the eye, so that scars have to be added to those already present, which is contraindicated cosmetically. When the surrounding skin is cicatricial it cannot be used for flaps. Every clinician will admit that the results of plastic operations with pedicled flaps are more certain and reliable. The operation of *Fricke*, in which a tongue shaped flap is formed with its base at the end of the loss of substance, is an extremely good one, but care must be taken that the base is broad enough, that it is not constricted too much, and that the subcutaneous fat is preserved with the vessels contained in it, as well as possible for the nutrition of the flap. I have had good results also from the cushioning of flaps with subcutaneous fat, according to the suggestion of *Silex* and *Axenfeld.*

Dieffenbach's operation is applicable chiefly to the lower lid when the loss of substance is in the form of a triangle with its base toward the margin of the lid. An incision is made prolonging the base of this triangle toward the temple, and from its end another is made downward, parallel to the outer side of the triangle. The flap thus marked out is dissected up and moved over nasally to cover the triangular loss of substance in the lower lid.

Blepharoplasty with Nonpedicled Flaps

The following points must be observed in order to successfully perform a blepharoplasty to restore parts of the lid, especially of the destroyed skin.

1. The margin of the lid is to be spared as much as possible in the excision of the cicatricial tissue, because the preservation of the palpebral fissure is absolutely necessary.

2. The cicatricial tissue must be removed as completely as possible, and the lid mobilized. All of the cicatrix, even in the subcutaneous cellular tissue, must be removed down to the margin of the orbit and the fascia tarsoorbitalis, for if it is left, and continues to contract, the flaps placed upon it will contract also.

3. All transplanted flaps contract, even when there is no subjacent cicatricial tissue; therefore a flap should be made at least a third larger than the space to be covered. When an operator has no measuring instrument devised for the purpose he can cut out the form of the defect to be covered in rubber tissue and lay out by this the flap to be taken.

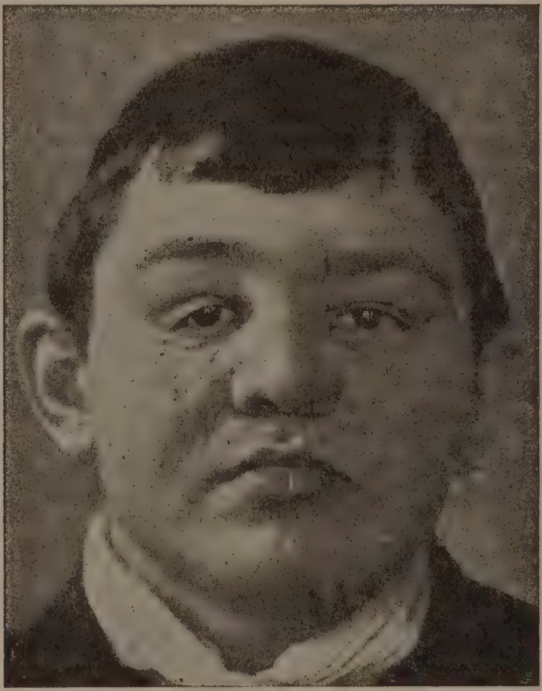


Fig. 61.—Plastic Operation for Ectropion Cicatriceum.
Right Eye: Nonpedicled Flap, Later Tarsorrhaphy of *Roemer*.

4. Usually it is necessary to suture temporarily the palpebral fissure in order that the transplanted tissue may quietly adhere. In this case we will take a large piece of the entire thickness of the cutis, because it contracts less, slide it over the bared surface, and slip its edges beneath the undermined edges of the defect. The dressing must be applied carefully without pressure. In grave cases such grafts must be renewed frequently and have to be repaired.

A great deal of patience is demanded, but finally both the surgeon and the patient are satisfied. The illustration shows the result of such an operation with a nonpedicled flap supplemented by a blepharorrhaphy.

76. Entropion and Its Treatment

This old man was operated on for cataract a few days ago. Normal healing was impeded by the fact that when the eyes were closed, especially under the bandage, the lower lid turned in so that the lashes and the skin rubbed upon the eye and caused irritation.

This other patient has trachoma, which has likewise produced an inversion of the margin of the lid. The lashes grow abnormally far backward and rub against the surface of the eye; they vary greatly in size: some are very fine, some like bristles, and are no longer arranged in a normal row, but are doubled in some places. In an entropion like this the lashes and the skin of the lid scour the eye; the defects thus caused in the epithelium of the cornea may become infected, and this mechanical irritation in trachoma often leads to pannus. The treatment of these forms of inversion must differ in accordance with the cause.

We understand by entropion an inversion of the free margin of the lid. It is caused in one of two principal ways, so we differentiate between an entropium spasticum and an entropium cicatriceum.

Etiology of Entropium Spasticum

In our first patient the margin of the lid turns in when the eye is closed, without any special spasmodic contraction of the orbicularis, and as no entropion can be produced in the healthy eye by the most spasmodic closure of the lids, it must be caused by something else.

The first factor in its production is a relaxed, folded condition, a loss of elasticity of the skin, a senile change. Spastic entropion is met with most frequently in old age and has therefore been called senile entropion, but it may also occur in other forms of blepharospasm in which the skin is relaxed, as in hysteria. The relaxed skin may be drawn along and the entire lid turned in.

The second factor is to be found in the contraction of the orbicularis. The palpebral portion of the orbicularis surrounds the palpebral fissure in such a way that the concavity of its arch is directed downward in the upper lid, upward in the lower, and the palpebral fissure is pressed together by the contraction of its fibres. In the tarsal portion of the orbicularis the concavity of the arch is directed backward and maintains the apposition of the lids upon the eyeball. So long as both of these parts are balanced the arrangement is most beautiful, but as soon as the bundles of fibres close to the margin of the lid preponderate the margin is pressed backward more strongly, is bent inward, and spastic entropion is produced. After an enucleation, when the lid has lost the support afforded by the eyeball, an entropion may readily be produced, which is known as entropium bulbale. In this case the bandage suffices to support the pressure with which the fibres of the orbicularis drags back the margin of the lid with the relaxed skin. In other cases there is to be added a loss of the elasticity of the tarsus and a forcing away of its upper convex margin from the eyeball by a swelling of the transition fold, particularly in the blepharospasm caused by eczematous conjunctivitis. The development of this form of entropion is also favored by a blepharophimosis.

Treatment of Entropium Spasticum

The right treatment cannot be given a case of spastic entropion until its cause has been ascertained. The entropium bulbale of anophthalmos is obviated by the introduction of a prothesis. In such a case as this one it sometimes suffices to draw down the skin of the lid frequently. The lower lid may be painted with collodion in order that when dry it may act upon the lax skin, or the lid may be drawn down by strips of adhesive plaster applied from close to the cilia downward and outward upon the skin of the cheek. If none of these measures are successful and the case is thought to be one of only temporary entropion, a thread may be introduced in the following way.

Take a suture armed with a needle at each end; introduce the first needle about 3 mm from the margin of the lid, pass it down beneath the skin and bring it out about 15 mm vertically below the point of insertion. Pass the second needle close to the first in the same way, so that the loop of thread lies on the skin near the margin of the lid. The ends of the suture are then to be tied over a small drainage tube, or a roll of cotton, with sufficient tightness to produce a slight degree of ectropion. The skin is shortened vertically by this enclosure of a fold by the thread. When the thread remains long enough, and it is necessary that it should remain several days, tense bands are formed along it which oppose the contraction of the orbicularis. Several such threads may be introduced to strengthen this effect. This operation suffices only in cases of transient, spastic entropion.

Another way in which the thread may be introduced is the *Snellen-Stellwag* method. Both needles are entered at the deepest place of the transition fold of the conjunctival sac and passed directly out through the skin, leaving a loop in the conjunctival sac parallel to the margin of the lid. The needles are then reintroduced in their wounds of exit, carried vertically upward between the skin and tarsus, brought out close to the outer edge of the lid and tied over a little roll. This operation can be performed only on the lower lid.

A horizontal fold of the lax skin may be excised in more obstinate cases. By means of the cicatrix the skin of the lid is shortened vertically and tension is made on its margin. The fold removed must not be too broad, or an ectropion may be produced. The part of the orbicularis nearest the margin of the lid may be excised to increase the effect and lessen the tension of this muscle.

In many cases the skin may be displaced horizontally in order to draw outward and backward the margin of the lid. A horizontal incision is made parallel to the margin, then two incisions converging downward from the ends of the first, and the included triangular piece of skin is excised from the middle of the lid. The two legs of this triangle are then sutured vertically. In cicatricial entropion the methods of procedure are entirely different.

Etiology of Entropium Cicatriceum

Every disease of the conjunctiva that results in cicatricial contraction may produce cicatricial entropion. The most common cause is trachoma, other causes are pemphigus and burns.

As the conjunctiva contracts the margin of the lid is drawn toward the eyeball, the inner edge is worn off and gradually the intermarginal space is obliterated. The cilia at first touch the eyeball only with their tips, but finally rub back and forth upon it with their shafts. Thus a pure entropion may occur in which the cilia are intact. Usually, however, the margin of the lid and the bed of the cilia are simultaneously diseased and entropion and trichiasis occur together. The margin of the lid itself is deformed, so we must examine every case to ascertain whether it is a case of pure entropion, or of entropion and trichiasis combined. The consequences of cicatricial entropion are the same as those of the spastic, only much more serious; pain, sensation of a foreign body in the eye, ulceration and pannus. The only treatment is operative.

Operations for Cicatricial Entropion and Trichiasis

Trichiasis is present in most cases of cicatricial entropion, and quite a number of operations have been proposed and recommended to obviate both conditions at the same time.

If there is little or no trichiasis *Hotz'* method is excellent. In this operation the skin is drawn up with all of the cilia and fastened to the tarsus, or the tarsoorbital fascia. When performed on the upper lid the first incision forms a curved line across the entire lid following the convex upper margin of the tarsus. This arched line may be seen on the closed lid as a wrinkle in the skin, which begins about 2 mm above the inner canthus, rises to the middle of the lid, and then falls away to the outer canthus. If the lid is drawn down hard this curved line is made straight and the knife may be drawn horizontally from a point 1 mm above the inner to 2 mm above the outer canthus. The upper margin of the wound recedes. We now cut through the orbicularis along the lower margin of the wound until the reddish yellow color of the tarsus comes into view, the upper margin of which is easily recognized. The tarsoorbital fascia need not be cut. The palpebral is thus separated from the orbital portion of the orbicularis. To bring the palpebral portion more plainly into view the lower margin of the wound is drawn down and then a strip of the muscle, about 3 mm broad, is excised from one canthus to the other with forceps and scissors. The margin of the skin and the upper part of the tarsus must be cleansed of all muscle fibres, and then the lower margin of the wound is sutured to the upper margin of the tarsus. The sutures are introduced through the lower margin of the wound upward, beneath the upper border of the tarsus through the aponeurosis, passed still higher to include the upper margin of the tarsus, and finally brought out through the skin of the upper margin of the wound. Fibres of the orbicularis must not be included in the suture. The threads are then tied. On the lower lid the incision is made about 3 mm from the free margin of the lid, and it is best to excise a fold of the skin at this place if it is superabundant. After the muscle has been abscised and the fascia tarsi laid bare, the sutures are introduced in the intermarginal space behind the cilia, carried beneath the fascia tarsi to the lower end of the tarsus, and passed out through the skin. When the sutures are tied the tarsus is stretched and the cilia are bent outward.

When trichiasis is associated with entropion I prefer the operation I learned from my former teacher *Vossius*. *Jaeger's* horn plate is placed beneath the upper lid, which is pressed firmly upon it to render it bloodless, while an intermarginal incision is made from the inner to the outer canthus. This incision is made of full length even when we have to deal with a partial trichiasis, for the results obtained after an incomplete intermarginal incision are not as good. In this way the margin of the lid is split into two laminæ, an anterior that contains the normal or abnormal cilia, and a posterior, which contains the tarsus and the *Meibomian* glands. This incision must be about 4 mm deep and must gape. Various methods of grafting have been tried to give the necessary breadth to the deformed and now gaping intermarginal space in order to turn the cilia away from the eyeball. Some ophthalmologists use mucous membrane from the lip, others have taken pedicled and nonpedicled flaps from the vicinity of the eye. If the flap is taken from the skin of the lid itself, the outer edge of the lid is dissected upward obliquely from both angles of the wound, for a distance of 5 mm, and then a flap of skin is formed broad enough to conveniently fill the gaping space, about 4 mm broad, by two parallel incisions along the margin of the lid. This flap is now dissected up until it forms a flap attached at each end, which may be shoved over the outer edge of the lid and laid in the gaping wound in the intermarginal space. The wound in the skin left by the removal of the graft is closed with sutures, the flap is carefully arranged in position, and fixed by numerous fine sutures to the anterior and posterior laminæ. I agree with *Vossius* that this operation complies with the cosmetic requirements; the scars are invisible, the flap heals faultlessly and prevents the orbicularis from turning the cilia again toward the eye. One advantage of this flap over grafts of mucous membrane is that it does not shrink so much. If a lash should grow in because its root has been cut it may be destroyed by electrolysis. This operation cannot be performed when the skin of the lid contains many fine hairs. The sutures may be removed after a few days during which the eye is kept quiet under a dressing of ointment.

77. Tuberculosis and Syphilis of the Eyelids

I. Tuberculosis of the Skin of the Eyelid

Clinical observation has shown that lupus rarely starts in the skin of the lid itself, but usually extends to it from the skin of the face. Lupus is one of the diseases that is not arrested at the margin of the lid, but extends beyond it. It is a disease of special interest to us because of the serious consequences produced by it in the lids. It causes great destruction of the skin and results in extensive cicatrization, which produces ectropion frequently. The ectropion is started primarily by the cicatricial contraction in the skin of the lid, but it is increased in many cases by the tension of the cicatricial skin of the face. Associated with the ectropion we meet with serious changes in the bed of the cilia, loss of the intermarginal space and trichiasis.

At the commencement of the disease rather brownish places, as large as the head of a pin, or of a hemp seed, are to be seen in the skin. These infiltrations may be flat (lupus planus), or elevated above the surface (lupus nodularis). If it is scaly it is called lupus pityriasiformis, or exfoliativus. As the disease progresses the redness disappears, and a small whitish area of contraction appears in the skin. When new nodules form about such cicatricial places we have lupus circinatus, or when the extension is more rapid and diffuse, lupus serpiginosus. As the lupus nodules break down ulcers are formed, which present a typical appearance, with sharp margins and bluish bases, covered in places by granulations; they are superficial and leave behind broad scars. One form, lupus disseminatus follicularis, exhibits a certain similarity to the nodules of acne.

Lupus is not the only form in which tuberculosis may present itself in the skin of the lid. Little nodules appear and in a comparatively short time break down into ulcers, the margins of which are rather red, but little infiltrated, and the bases of which are covered with soft, yellowish granulations. New nodules appear in the vicinity, soon break down and cause an extension of the ulcer. A favorite seat of such ulcers is the inner half of the lower lid. In such a case we have to notice whether the disease is a primary one of the lid, or secondary to a tuberculous ulcer of the conjunctiva tarsi, for the tubercle bacilli from the conjunctiva may infect the macerated skin, and such ulcers may occasion serious changes in the margin of the lid. Tuberculous ulcers should be curetted with a sharp spoon and be dressed with iodoform.

The eyelids suffer from tuberculosis still more frequently when the pus finds its way through the lid and establishes a tuberculous fistula from a tuberculous periostitis of the margin of the orbit. Such fistulæ are most frequent in connection with tuberculosis of the lacrymal sac. The long duration is characteristic, the fistulæ are filled with spongy granulations and new infections may extend to the neighboring skin. The fistulæ and the lacrymal sac should be carefully extirpated, the diseased bone removed and, when necessary, a plastic operation performed. Tuberculous fistulæ very often cause ectropion. Lupus, tuberculous ulcers and fistulæ are the commonest forms of tuberculosis in the eyelid. Scrofuloderma is observed much more rarely in the lids of children. This disease begins as a movable nodule as large as a hazel nut or bean, which breaks down in the middle, ruptures and evacuates some pus, leaving an ulcer within which are granulations that contain tubercle bacilli. Equally rare are the chalky tuberculous nodules in the subcutis of the lids which are perfectly unirritating. Their nature is learned first from anatomical examination.

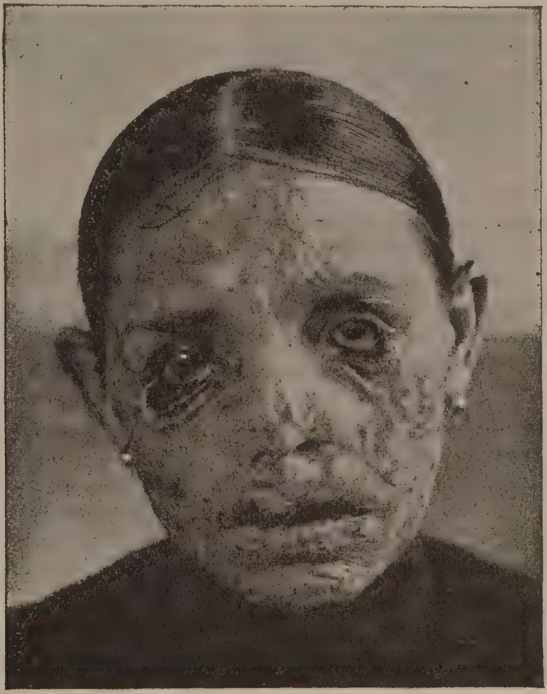


FIG. 62.—Ectropium Cicatriceum Caused by Lupus of the Face.

II. Syphilis of the Eyelid

I shall mention briefly the essential points of *syphilis of the eyelid* not only to round out our studies of the diseases of the lids, but also to urge the need of care in the treatment of syphilitics. It has happened repeatedly that physicians have been inoculated with syphilis while engaged in the practice of their profession, and have had chancres of the eyelid develop from mucus coughed into their eyes while they were making applications to the throats, or larynges of patients. Other methods by which the lids are infected are by kissing, by the use of infected handkerchiefs, by vicious customs, such as licking sore eyes with the tongue, and sexual perversions. All the characteristic manifestations of the primary, secondary, or tertiary forms of syphilis may be met with on the eyelids. The symptoms of hereditary syphilis on the lids resemble in many ways those of the acquired disease, so it will suffice to speak only of the latter.

1. *Chancre of the Eyelid*

Chancres are usually met with in the intermarginal space of the lid, and are rather difficult to recognize at first. A chancre starts as a dense infiltration, a reddish papule, and the attention of the careful observer is finally attracted by its obstinacy and peculiar hardness. The thickening of the margin of the lid increases, the epithelial covering is thrown off and the erosion begins to weep; the skin of the lid becomes swollen and crusts begin to form. The surface of the erosion bleeds easily, or appears peculiarly moist and lustrous, while the infiltration increases, the tissue surrounding the ulcer is as hard as cartilage, the margins of the ulcer are wall like, its base has a dirty coating and the nature of the primary sore can no longer be mistaken. *v. Michel* has pointed out that several infiltrates that break down into pus may be formed in the vicinity of the ulcer. When the crust is removed from such an ulcer the base looks peculiarly lardaceous. A second chancre may develop in the corresponding place on the other lid. The swelling extends, the inflammatory irritation causes a great increase of the conjunctival secretion and the clinical picture becomes threatening.

About two weeks after the appearance of the primary lesion a slow, painless swelling of the lymphatic glands in the vicinity forms an evidence of the commencement of the general infection. It has been observed frequently that when the chancre is in the middle of the lid the submaxillary gland swells first, when it is in the outer portion the preauricular gland is first affected; this corresponds to the division of the lymphatic vessels in the lid. The chancre may spread to the conjunctiva as the infiltrated tissue breaks down. A hard scar remains after the ulcer has healed and a permanent partial alopecia may be left, but the lashes grow again in many cases. Timely specific treatment is naturally important.

2. *Secondary Stage of Syphilis of the Eyelid*

Papules are apt to develop on the covering fold of the skin of the upper lid in the secondary stage of acquired syphilis, especially near the outer canthus. When the papules are small, nodules of the size of a pin's head are arranged in groups, or circles, when large the nodes are larger. They are of a peculiar copper color, but when they exfoliate the color is changed to a dirty yellow, and the condition is then called a papulosquamous syphilide. A brown spot remains for a long time wherever a papule has been situated. The diagnosis is easy when this exanthem is present elsewhere on the skin, but it sometimes happens that the skin of the eyelid is the only place where the eruption appears. The eyelashes and eyebrows not infrequently fall out with the onset of the secondary symptoms.

3. *Tertiary Symptoms of Syphilis of the Eyelid*

Firm, brownish, smooth nodules may appear on the margin of the lid in the tertiary stage of syphilis. Such a node is a superficial gumma, or a tertiary papule. It is usually obstinate, though it may be resorbed after causing the cilia to fall out, or it may break down and give rise to an ulcer which spreads and destroys a large part of the margin and skin of the lid. A gumma may also be situated in the deeper layers of the subcutis, where it forms a large, roundish node, which may break down in a very short time, causing a deep ulcer associated with severe inflammatory symptoms. The differential diagnosis between this and a tuberculous or a carcinomatous ulcer may be difficult. In a tuberculous ulcer the soft granulations must be noticed, in carcinoma the destruction of tissue is much slower than in a softened gumma; in doubtful cases the effects produced by large doses of potassic iodide may be awaited and above all recourse should be taken to *Wassermann's* reaction.

78. Malignant Tumors of the Eyelid

This patient, 73 years old, has had a malignant growth on his right lower lid for two years. It developed gradually and has reached its present size in spite of the caustics, curettage and irradiation employed by his physician. It began as a small, hard node; now the entire lower lid, from one canthus to the other, has been replaced by an ulcer covered with crusts, which bleeds easily and excretes pus. The ulcer involves the lower transition fold and even a part of the conjunctiva bulbi. Its margins are thick, elevated and very hard; its surface is irregularly rough and granulating. No trace of the caruncle can be seen at the inner canthus, where a piece of the skin as large as a cent has also been destroyed. The eye has become very irritated because the absence of the lower lid renders closure of the eye impossible. From the age of the patient, the beginning and course of the disease and the nature of the ulcer it may be correctly surmised that this is a carcinoma of the lid. The lymphatic glands in the vicinity are intact in this case.

The malignant tumors met with on the eyelids are carcinoma and sarcoma.

I. Carcinoma of the Eyelid

The commoner form is carcinoma. It usually appears on the lower lid and by preference at the inner canthus. Its progress is extremely slow. A clinical differentiation may be made between a superficial and a deep form. It usually starts as one or more hard nodules on the margin of the lid. As the superficial form extends the epidermis is thrown off in one or more places so as to form erosions that are moist at first, but soon become covered with crusts and scabs. A true cancerous ulcer gradually develops from the erosion through infiltration and necrosis. The base of the ulcer is irregularly segmented, one part giving rise to papillary excrescences while in another cicatrization accompanies the extension of the cancerous infiltration. Firm connective tissue is formed and the healthy skin of the lid may be thrown into folds by the cicatricial contraction. Such cancerous ulcers not rarely become skinned over with epidermis, but healing does not follow. The margins in this case are studded with hard nodules. In the deeper form the process is more rapid. The nodes in the margin and floor of the ulcer spread; the cancer involves the neighboring skin and the eye itself is in danger. In this patient the carcinoma has involved the palpebral conjunctiva, destroyed the entire lid, even the bulbar conjunctiva is no longer intact below, and the danger to the cornea from desiccation and ulceration in such a lagophthalmos hardly needs to be pointed out. A carcinoma in the vicinity of the eye may involve the lid secondarily. Other points in the clinical picture of cancer of the lid are that it is met with only in old age, usually between the ages of 50 and 70, that the lymphatic glands in the vicinity are very rarely involved and that it shows no tendency to metastasis. So far as life is concerned this form of carcinoma may be called benign.

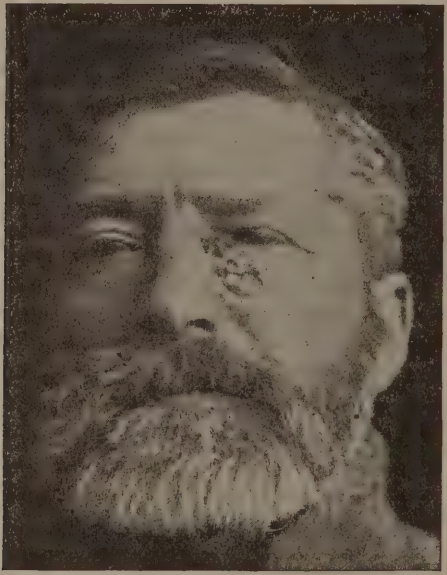


FIG. 63.—Carcinoma of the Lid and Skin of the Cheek.

This benign nature of carcinoma of the lid has led to many attempts to cure it without operation, by means of cauterization and such caustics as zinc chloride, lactic acid and arsenical paste. Recently treatment with the X-rays and with radium has been recommended and tried a great deal, but, aside from the fact that the therapeutic use of these chemically acting rays is not easily applicable to the eye, I must say that the results that have come under my observation are not very satisfactory. This patient has been treated in such a manner for a long time. I can say nothing of the brilliant results that are frequently reported, but I would not advise much time to be lost in trying these methods. After its trial, as before, treatment consists only in the radical, surgical extirpation of the tumor.

Histologically, carcinoma of the lid may be either an epithelioma with characteristic epithelial pearls, or an adenocarcinoma which originates from the glands of the lid. We find among them tumors which show only a slight anaplasia of cells and only a slight depth, and others with very marked anaplasia and extremely multiform cells. The adenocarcinoma has a glandular construction in which cornification is absent. Usually the epithelial cones can be seen to penetrate in great numbers and can be sharply differentiated against the connective tissue portion of the skin. They contain processes of softening and signs of necrosis.

Besides carcinomata we find in the lids tumors which present the same clinical picture, but are recognized microscopically to be endotheliomata, from the predominance of the proliferated, endothelial cells. They likewise occur only in old age.

II. Sarcoma of the Eyelid

Sarcoma may be clinically distinguished from carcinoma of the lid first by its malignancy. It is met with at all ages. *Samelsohn* reported a sarcoma of the lid in a child ten months old. On the whole these tumors are rare. The commonest form is the round celled variety which at first greatly resembles chalazion, but its rapid growth and the necrotic destruction of its interior, sometimes with hemorrhages, soon reveal its nature. A round celled sarcoma consists of narrow bands of connective tissue with cells that resemble lymphocytes with large nuclei heaped between them. Next in frequency comes the melanosarcoma of the lid, which often starts from a pigmented nævus. The diagnosis of the gray, or black brown, tumor presents no difficulty. If the tumor is situated deeply it appears gray black through the conjunctiva. Not infrequently it springs from the connective tissue between the fibres of the orbicularis. Spindle celled sarcomata and myxosarcomata are much more rare. Among the forms met with on the lid are the plexiform angiosarcoma and cylindroma, in the construction of which other tissues are involved. These are not pigmented and sometimes the diagnosis may waver between gumma and malignant tumor. Finally it may be mentioned that a recurrent glioma may spread to the skin of the lid and involve the parotid. Radical extirpation at the earliest possible moment is indicated in all of these malignant tumors.

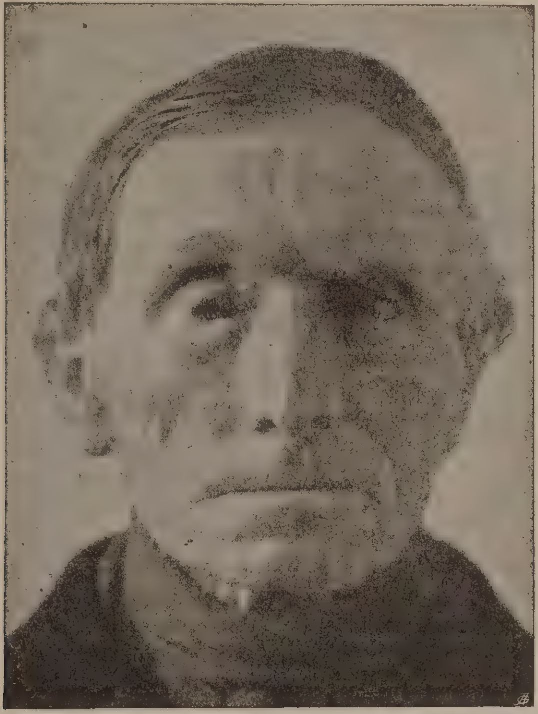


FIG. 64.—Plastic Operation on the Lid. Restoration of a Lid Destroyed by Carcinoma by Means of a Pedicled Flap Taken from the Forehead.

We first remove all of the diseased portion with enough of the neighboring tissue to enable us to hope that we are in healthy tissue, which leaves a large defect. The lower lid, the lower transition fold and even a part of the conjunctiva bulbi have been removed, as well as the entire inner canthus. This defect is now covered with a broad pedicled flap of considerable size taken from the forehead. This flap must be well nourished by the vessels of the inner portion of the brow and of the bridge of the nose. It is now in place, lies well, covers the entire defect and its lower portion is carefully secured by sutures. Although the conjunctiva bulbi has been partially removed we try to save the eye with normal vision. It is to be expected that the upper margin of the flap, which is not sutured, will shrink downward and roll in somewhat, so that it will gradually leave bare the anterior surface of the eye. The raw surface left in the forehead by the removal of the flap is now covered by drawing the skin together with sutures, and by applying *Thiersch* grafts taken from the arm. After the flap has healed well in position the bridge is divided and the swelling produced on the bridge of the nose is removed.

I present here a patient on whom this operation was performed after an extensive carcinoma of the lid had been removed. There is no recurrence and he can use his eye, although the conjunctival sac is very small.

79. Benign Tumors of the Eyelid

A number of brown, yellow, or clay colored spots covered by normal epidermis may be seen in the skin of both the upper and lower lids of this patient. Only a slight consistency can be felt in the skin. These are superficial little tumors, situated principally at the inner canthus of each eye, which are neither sarcomata nor carcinomata, and therefore not malignant.

The benign tumors of the lids are rather numerous and their division into congenital and acquired is of assistance in differentiating them. A glance at this chart will help in the diagnosis of each case. The question whether the tumor is congenital or has existed since early childhood is readily answered.

Benign Tumors of the Eyelid

I. Congenital	*II. Acquired*
1. Angiomata.	1. Warts.
2. Lipomata.	2. Cornua Cutanea.
3. Dermoids.	3. Fibromata.
4. Nævi.	4. Xanthelasmata.
5. Neurofibromata.	

I. Congenital, Usually Benign Tumors

1. *Nævus of the Eyelid*

Two forms of nævi are met with on the eyelids: nævi spili, smooth, soft spots of pigment, and nævi pilosi, hairy formations resembling warts which are sharply elevated above the surrounding surface. Pigment spots usually are stationary, or have only a slow growth, but their extirpation is indicated because clinical experience has taught us that eventually they may give rise to malignant sarcomata. A pigmented nævus consists of nævus cells, derived partly from the epithelium, partly from the connective tissue cells, which lie heaped together with pigment cells, the heaps separated by a retiform connective tissue.

2. *Angioma of the Eyelid*

These, which are easily recognized by their color, are divided into hæmangioma simplex and cavernosum. The simple angioma consists of neoplastic, or of old, dilated blood vessels with thickened walls. They lie partly on the surface, partly in the subcutis and form dark red spots, or roundish tumors which are usually sharply defined and larger. When a child with such an angioma cries the tumor may be seen to swell. The margin of the lid may be affected as well as its surface and other angiomata may be present elsewhere in the body. When such a tumor is compressed it becomes smaller, but regains its size as soon as the pressure is removed. Although it is not malignant it may grow very large and give rise to elephantiasis hæmangiectatica, or teleangiectodes. The cavernous angiomata are those which have large hollow spaces filled with blood; they may be little blue growths, or large, lobed tumors, and they swell whenever the return flow of the venous blood is obstructed. They grow slowly, but may reach an enormous size (elephantiasis cavernosa). Treatment consists of extirpation. In small growths I have obtained good cosmetic results from punctate cauterization with the electric cautery. I have had no experience with subcutaneous injections of alcohol in their treatment. The method of *Payr*, to treat these tumors by studding them with little magnesium points, is applicable only to cavernous angiomata, as he has always claimed. Lymphangiomata of the lids sometimes attain an enormous size; these are congenital and consist of hollow spaces, the walls of which are lined with a single layer of epithelium, have a pale appearance and resemble cysts. With increased growth lobulated tumors are formed which are known as elephantiasis congenita lymphangiectatica.

3. *Lipoma of the Eyelid*

Lipomata are rarely met with in the lids. They form elastic tumors which frequently are enveloped in a connective tissue capsule and may extend more or less into the orbit.

4. *Dermoids*

These form firmly attached, spherical growths in the skin of the lid, situated, as a rule, where sutures and fissures existed during fœtal life, as at the inner canthus, where the frontal process unites with the maxillary process, or laterally at the upper margin of the orbit, at the zygomatic frontal suture. Removal of the tumor not infrequently reveals a diastasis of the suture. Sometimes they are attached to the periosteum by means of a pedicle. The skin covering them is unirritated and movable. During extirpation of these tumors the operation must often be carried far into the orbit. A dermoid consists of a thick wall that encloses hairbulbs, sebaceous glands and sweat glands.

5. *Neurofibroma of the Eyelid*

Neurofibroma, the last congenital form of tumor of the eyelid, is divided by *v. Michel* into the plexiform neurofibroma, the fibroma molluscum and the hypertrophy of one side of the face. The plexiform neurofibroma is characterized by the fact that it always primarily affects the upper eyelid, in which hard cords that wind about and join together may be felt. These are also called vermiform, or bulbous growths. Buphthalmos is not infrequently met with on the same side. The tumor consists of numerous tufts of nerves enveloped by a dense connective tissue capsule. The proliferation of the connective tissues comes from the perineurium. It is remarkable that such a tumor is painful only in certain places. In fibroma molluscum the lid is the seat of a soft, pedicled tumor, usually associated with similar mollusca of the skin of the face and neck, in which the fibromatous proliferation starts from the sheaths of the subcutaneous nerves. In congenital hypertrophy of one side of the face there is a sharp line of demarcation between the hypertrophic and the normal sides. The hypertrophic side is markedly thickened and feels soft, while the upper lid is enormously enlarged and droops very low. This condition is frequently met with in combination with the plexiform neurofibroma and is almost always associated with buphthalmos. This disease is a proliferation of the perineurium and endoneurium of the nerves of the skin, together with a diffuse fibromatosis of the skin and of the subcutaneous cellular tissue. Fibromatosis of the ciliary nerves plays a part in its origin.

II. Acquired Benign Tumors of the Skin of the Eyelid

1. *Warts*

The verruca vulgaris, verruca plana juvenilis and verruca senilis have all been observed on the lids. The common wart is a hard papillary growth with a surface that is rugous, or full of clefts. Anatomically it consists of an elongated papilla with proliferation of the stratum spinosum and thickening of the epidermis. The verruca plana is a flat, yellowish brown elevation met with in the skin of the lid and forehead particularly of young people. In old people yellow brown warts covered with horny scales are met with in the skin of the face and lids. These are of more importance than the other varieties because they may give rise to carcinomata.

2. *Cornua Cutanea*

Horny outgrowths of the skin are rarely met with on the lids. They consist of proliferated cells of the epidermis with particularly marked cornification. They are striated either transversely or longitudinally, are of a dirty gray or brownish color and are formed only in old age. Both these growths and warts should be abscised and their bases cauterized. Acanthosis nigricans is a rare disease in which numerous thick papillary growths appear on the surface of the lid, or in the intermarginal space, at the inner canthus and may be associated with a gray yellow, or brown, pigmentation of the skin of the face.

3. *Fibromata*

Fibromata are hard tumors that hang from the skin by slender pedicles. They are usually small and have been compared to little hanging bags. They may form larger nodes in the subcutaneous tissue, in which they are usually enveloped in a capsule. The clinical picture presented on the eyelids by hæmangioendothelioma tuberosum multiplex, which is also known by other names, is very rare. Numerous little reddish yellow nodules, of about the size of the head of a pin, lie scattered in the skin, with which they are movable, and are surrounded by fine, reddish areolæ formed of ectatic vessels. These little growths are benign and do not exceed a certain size.

4. *Xanthelasma*

We come now to the form of tumor met with in our patient. Xanthelasma is a growth in the skin of the lids that develops, mainly in middle aged and old women, about the inner canthus. The little tumors may be scattered about the entire eye. There are two varieties, xanthelasma planum and tuberosum; the latter is rare. The growth of these tumors is very slow and causes no pain. As a rule the newly appearing spots are separated from the others by normal skin. The cause is unknown. Anatomically they consist of heaps of certain cells, called xanthom cells, in the form of nests. The xanthom cells are filled with fat granules and are derived from embryonal fat cells, or from the perithelium of the vessels. The idea that xanthelasma is a hyperplastic development of sebaceous gland cells is incorrect. These tumors are removed only for cosmetic reasons.

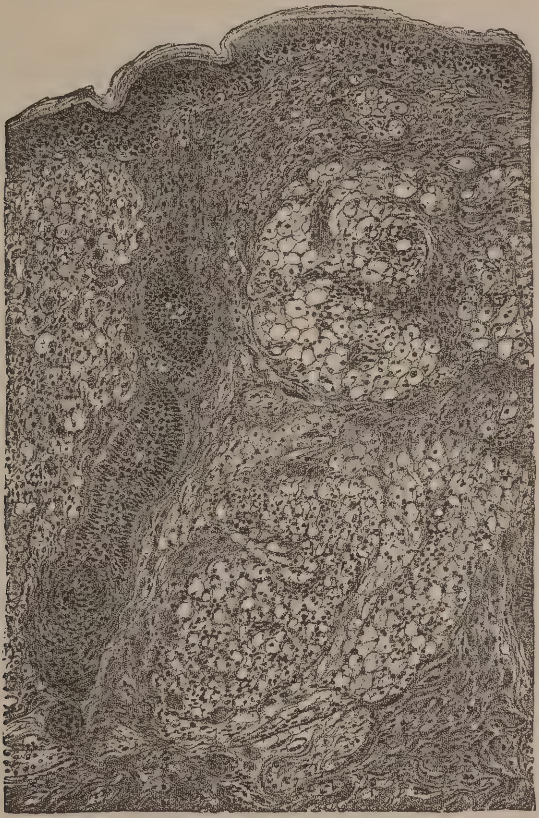


FIG. 65.—Xanthelasma.

Thus it may be seen that though the benign tumors of the lid are many, each variety has its own peculiarities, and that it is not difficult, as a rule, to make a differential diagnosis between them.

80. Abnormal Discolorations of the Skin of the Lids and Their Diagnostic Signification

In farmers, who work in the open air, in wind and rain, and are exposed to the rays of the sun in summer, the skin of the lids takes part in the diffuse pigmentation. We suppose that the chromatophores of the skin are increased by the action of certain light rays. Where the covering fold of the upper lid covers a part of the skin the pigmentation is less. This kind of pigmentation, which depends on the action of light, we call chloasma caloricum.

This child furnishes an example of the well known freckles: little, pale yellow, or dark spots in the skin of the face and lids, which grow dark with the advent of spring and pale again in winter. The action of light is not their sole cause, many families are predisposed to them.

This other patient, a pregnant woman, presents the well known chloasma gravidarum; bright or dark brown spots lie on both sides of the forehead and extend from close to the margin of the hair down upon the cheek, including the eyelids. In such a case the discoloration of the skin of the lids may be of diagnostic value, especially as anomalies of pigmentation may occur in the eyelids in quite a number of diseases.

We meet with a similar chloasma of the lids in the course of serious cachectic conditions due to carcinoma and tuberculosis, but it is of diagnostic importance mainly in exophthalmic goitre, *Addison's* disease, chronic arsenic poisoning and, in rare cases, ochronosis.

A marked pigmentation of the skin of the lids may occur in *Basedow's* disease, or exophthalmic goitre; in *Addison's* disease the lids, especially their margins, have the same bronze discoloration as the rest of the skin. The involvement of the lids is less in the pigmentation of the very rare ochronosis. It need hardly be mentioned that in jaundice the skin of the lids partakes of the general yellowish green discoloration.

The internal use of silver may also cause a peculiar, pale, bluish stain of the skin of the lids from the deposit of silver in the uppermost layers of the epidermis and the membrana propria of the sweat glands. In argyrosis of the conjunctiva also the skin of the lower lid particularly may become discolored.

Lack of pigment, the opposite of hyperpigmentation, is met with congenitally in albinism; the lashes and eyebrows may be white, while the skin of the lid is perfectly clear.

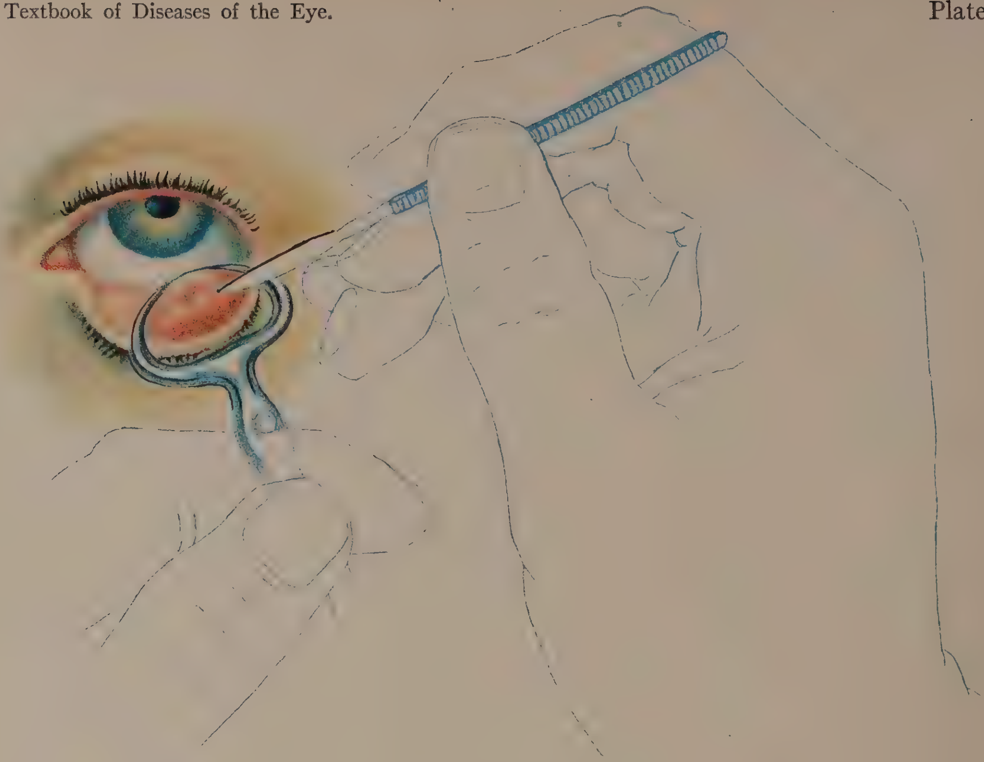
Vitiligo is an acquired decolorization of the skin which may appear on the lids in the form of clear, whitish spots and may also decolorize the cilia.

Xeroderma pigmentosum is a rare affection of the lids in which a peculiar piebald appearance may be given the skin by an abnormal pigmentation with simultaneous cicatrization. A part of the skin is dry, with numerous spots of pigmentation elevated above its surface, to which are added teleangeiectasies, and wartlike formations of a dark brown color. These may finally give rise to malignant growths. Xeroderma develops in children during the first year of life and is reported as a family appearance. The physician has little to do with such rarities, but the diagnostic signification of the other discolorations of the skin of the eyelids is useful.

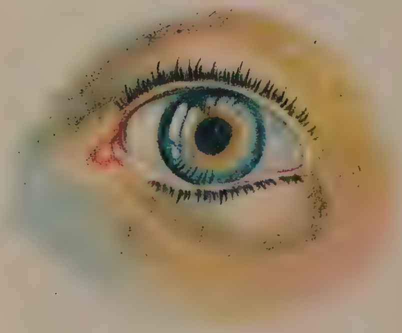
81. Atrophies and Hypertrophies of the Skin of the Lid

I. Atrophy of the Skin of the Lid

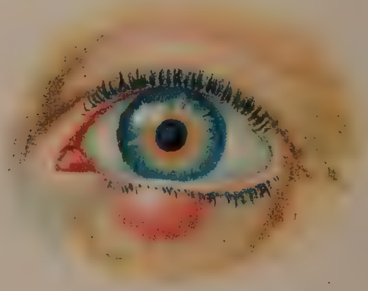
The characteristic feature of the clinical picture presented by this young man is that the skin of both upper lids has become so thin that it has fallen into numerous fine folds, a condition that has been compared to wrinkled silk paper. Numerous dilated vessels shine through this thin skin and in consequence of its atony the upper lid hangs down almost like a reddened bag and simulates ptosis, but the margin of the lid is not lower than it should be. This



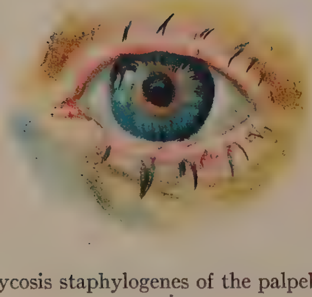
a Removal of a chalazion.



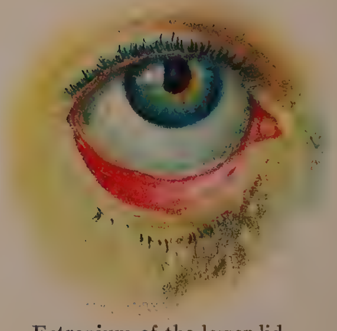
b Chalazion.



c Hordeolum externum.



d Sycosis staphylogenes of the palpebral margin.



e Ectropium of the lower lid.

case presents an advanced stage. The blepharochalasis usually affects young persons. It begins with an attack of inflammatory œdema of the upper lids, which disappears after a short time and then recurs. This peculiar atony of the skin develops gradually. *Fuchs* has called attention to the fact that the superficies of the skin is increased; when we seize the cilia of the upper lid, draw it down and measure its extent the enlargement of the surface in blepharochalasis is evident. The tarsoorbital fold especially is involved. The origin of this disease is still unknown. The transient œdema may be the expression of an angioneurosis. Excision of the redundant skin has been recommended, and *Fuchs* advises to fasten the skin to the upper margin of the tarsus by means of *Hotz'* operation in order to limit the drooping.

This is a case of atrophy confined to the skin of the upper lid. Another form is one of the symptoms of hemiatrophia facialis progressiva, or neurotic facial atrophy, a trophoneurosis which arises from the trigeminus, the *Gasserian* ganglion, or the sympathetic. The disease consists of a partial, or total atrophy of the constituent tissues of one side of the face, in which both the skin and the subcutaneous cellular tissue of the lid become atrophic.

A third atrophy of the skin of the lid is one of the symptoms of a general atrophy of the skin. Such an atrophy may be congenital in rare cases, because of an aplasia of the constituent tissues of the skin.

The general atrophy of the skin in old men can be recognized from afar. This senile atrophy is particularly marked in the lids; the skin is withered, wrinkled and frequently yellowish. The eyeballs are deeply seated, showing that the orbital cellular tissue is likewise atrophic. We also meet with such an atrophy of the skin in cachectic conditions, as well as in cretinism.

II. Hypertrophies and Degenerative Changes in the Skin of the Lid

Among the hypertrophies of the skin of the lid may be enumerated acne rosacea, scleroderma, elephantiasis nostras and rhinophyma. Various processes take place in the diseased skin in acne rosacea which give rise to nodes, pustules and a thickening of the skin that may form the source of large outgrowths. The lid is involved only by extension to it of the disease.

The skin of the lid may become thickened after repeated attacks of erysipelas, or eczema, and then the upper lid may be transformed into a thick, drooping swelling of a bluish red color. Changes in the blood and lymphatic vessels are responsible for this condition. Excision of the thickened part of the skin must be tried in such cases as these.

In scleroderma we have sometimes a circumscribed, sometimes a diffuse thickening of the skin of the lid. At first the skin is œdematous, then it hardens and finally atrophies. Scleroderma belongs to the diseases of the thyreoid gland.

In myxœdema the lids are swollen and protrude like sacs, the skin is pale and cool.

The so called colloid milium forms a flat, nodular elevation in the skin of the lid and may be recognized by its striking transparency.

82. Epicanthus and the Congenital Anomalies of the Lids

In operations on the eyelids we have dealt principally with acquired changes in their form and position. It may be added that occasionally the outer canthus may gape so as to allow us to look into a pocket formed of conjunctiva near the eyeball. It also happens in old age that pouches form in the lids over which the skin is very thin. According to *Merkel* this change is due to the fact that in old age all the fasciæ, including the fascioorbital septum, is stretched on account of atony.

Schmidt-Rimpler has described a hernia of fat in the upper lid which came through a defect in the fascia and a separation of the fibres of the orbicularis.

Finally the covering fold becomes greatly developed in old people with relaxed skin and wrinkles. With us the transverse fold of the upper lid hangs almost always from the demarcation between the orbital and the tarsal portion.

The commonest congenital anomaly of the lids is the fold of skin which partly covers the inner canthus on each side of the bridge of the nose of this child. This malformation, known as epicanthus, is frequently associated with other congenital faults of the eye. A slight degree of epicanthus that disappears as the body grows is seen not rarely in infants. If the epicanthus persists, the bridge of the nose appears to be markedly broadened and there is a superabundance of skin on the glabella. Strabismus may be simulated by an epicanthus, but is really present in many cases. The epicanthus may be partly corrected by lifting up the skin over the bridge of the nose, so *v. Ammon* suggested the excision of an oval piece of skin at this place, but this operation does not give good results; it is better to remove the folds themselves. Epicanthus is divided into superciliaris, palpebralis and tarsalis according to the place whence the fold springs.

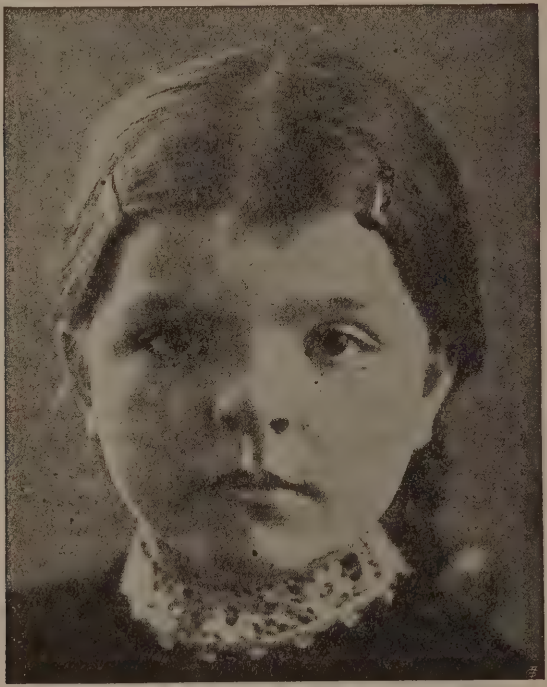


FIG. 66.—Epicanthus.

Another congenital anomaly of the lid is the coloboma, by which is understood a cleft, usually of a triangular form with its base toward the margin of the lid. It is met with most often in the upper lid, usually in its middle or in its inner segment. The margin of the cleft is covered by conjunctiva. A dermoid of the cornea is coincidently met with not infrequently. Colobomata of the lid are not uniform in their origin. The beginning of the lid can be recognized embryologically in the third month, when swellings of the skin appear above and below the eye, grow toward each other and unite. After this occlusion of the palpebral fissure the formation begins of the constituents of the margin of the lid, the cilia and glands, and coloboma may be considered as in part due to inhibition of development. Another view is that a coloboma is the result of early synechiæ between the surface of the embryo and the inner wall of the amnion. According to *Kraske* a coloboma at the inner canthus is connected with the oculonasal fissure. The margins of a coloboma may be freshened and united.

Other malformations of the lids are of little interest. The lids may be abnormally short; the highest degree of this abnormality is total absence, ablepharia. When the palpebral fissure remains closed the condition is called cryptophthalmos; the eyeball may be either normal or undeveloped. Sometimes the margins of the upper and lower lids are united by only a thin cord, ankyloblepharon filiforme.

VI

Injuries of the Eye

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83. Frequency and Causes of Injuries of the Eyes and the Natural Devices for Their Protection

We may refer to the statistics of the Imperial Insurance Department to learn the frequency of injuries of the eye in comparison with injuries of other parts of the body. There we find that in 1887 the eyes were injured in 914 out of a total of 15,970 accidents in factories, 5.73%, and that in 19,359 accidents in the country the eyes were injured in 806, 4.14%. In some trades the percentage is higher; it ranges from 4 to 10%. In many trades there is one serious injury of the eye yearly among every 1,000 workmen and one injury to the eye in every ten accidents. But these figures include only comparative serious injuries of the eye; the total number is much greater. The lodgment of little foreign bodies in the conjunctival sac is of daily occurrence; the majority of such patients do not seek a physician, they remove the foreign body themselves.

In our clinic, injuries of the eye form from 6 to 12% of the entire clinical material. Men suffer far more frequently from injuries of the eye than women, and the laboring class is the one most exposed to the danger, but it is a very remarkable fact that a large percentage of the serious injuries of the eye occur in childhood. It is also well to bear the fact in mind when called upon to pass judgment on an injury of the eye that if morbid conditions already exist in an eye the danger of injury, both as to liability and results, are increased. For example: A near sighted person, or one with impaired vision, does not instantly see the threatening danger, and an insignificant injury to an eye that is not normal may have disastrous consequences. Thus detachment of the retina has often been observed to follow slight injuries in myopic eyes; rupture may be caused more easily in an eye with ectasia of its capsule, as in buphthalmos or staphyloma, than in a normal eye; when vascular changes are present hemorrhages are more frequent and more easily caused, and congenital or acquired relaxation of the zonule favors the production of a complete luxation of the lens.

It has been proved by a large mass of statistics that neither eye is endangered more than the other, except that in metal workers and stone cutters the left eye seems to be the more exposed. Injuries of both eyes are met with frequently after explosions and burns. These statistics show that the *causes* of injuries of the eye are very numerous.

Many callings predispose directly to certain injuries of the eye. In the locality of iron and steel industries, tinsmiths and fine mechanical arts, injuries from splinters of metal, ashes and molten metal are very apt to be met with. Miners and stonebreakers suffer particularly from injuries by foreign bodies. Powder and dynamite explosions are also to be taken into account. In masons and carpenters we think first of lime burns, splinters of wood and stone. The chemical industry furnishes burns with acids and corrosive agents. In the fields and forests superficial abrasions, which lead so readily to ulcus serpens, come to the front, though injuries from the horns of cattle, or kicks of horses, are not uncommon.

Next to the occupational dangers comes the unlucky accident. Examples need not be quoted. Thoughtlessness and carelessness will never cease to destroy human eyes. Think of the thoughtless handling of firearms reported daily in the newspapers.

Grave self inflicted injuries and mutilations of the eye, practiced in some countries by people who wish to avoid military service, are hardly met with among us, except in lunatics. Malingerers frequently place irritating substances and foreign bodies in the conjunctival sac in order to give plausibility to ascribed troubles.

A conception of the social importance of injuries of the eye may be obtained from figures that show how frequently such injuries are the cause of blindness. *Magnus* found injuries to be the cause in 8.05% of 3,204 children blind in both eyes. The percentage is given as greater in other statistics, 10%, 20% and upward.

Another means by which we may measure the consequences of injuries of the eye is the number of enucleations after traumatism. Statistics show that from 50 to 70% of all enucleations are performed because of injuries and of their consequences.

The frequency with which the eye is injured is not surprising when we take its exposed position into account; the frequency would be much greater if the eye were not provided with a number of natural means of protection. The eyebrows and the overhanging upper margin of the orbit protect the eye from injuries coming from above; the lids with their lashes intercept many foreign bodies and insects; above all, the eye is protected by its situation in the orbit from injuries coming from the sides and behind. The firm bones of the margin of the orbit intercept many blows, but those of the lower and outer margin do not project as far forward as those above and to the inner side.

The spherical form of the eyeball and its position in the orbit is another important factor. The eye can give way backward and to the side while the cushion of fat mitigates the force of the blow. The eyeball may also be displaced forward somewhat without injury, because the muscles of the eye can be stretched and the optic nerve is rather tortuous in its course through the orbit. The eye consists of a tense, strong capsule and partly fluid contents, with an intraocular tension of 24 mm of mercury, therefore a greater pressure than this is needed to stretch and lacerate the tissues within it.

The reflex closure of the eyes and winking form still another means of protection. When the approach of a foreign body is perceived the eyes close involuntarily, while many little foreign bodies are removed by winking. An increased secretion of the tears is also excited as a rule, which aids in their removal, dilutes injurious substances and lessens their effect.

84. Superficial Injuries of the Eye

For practical reasons we divide injuries of the eye into three large groups: I, superficial injuries; II, contusions; III, perforating wounds. Usually the group to which any particular case belongs may be determined from the history.

In superficial injuries the violence is slight and the lesions are on the surface; in contusions the eye has been struck by some blunt object; in perforating wounds the capsule of the eyeball has been penetrated by a more or less sharp object. When a patient with an injury of the eye enters the office it is necessary to determine to which class the injury belongs in order to treat it properly.

Superficial injuries include lesions of the conjunctiva and of the outer layers of the cornea. Three forms come into consideration:

1. Foreign bodies in the conjunctival sac and on the cornea.
2. Nonperforating injuries and erosions.
3. Burns.

I. Foreign Bodies in the Conjunctival Sac and on the Cornea

It is a daily occurrence for a patient to have something fly into his eye; the eye becomes photophobic, painful, lacrymates and the conjunctiva becomes injected. A characteristic feature of this condition is that the eye is less painful when kept quietly closed. Little particles of coal, cinders, wings of insects and grains of sand lodge on the surface of the eye and are often wiped away by the movement of the lid. If they lie beneath the lower lid they cause little trouble, but beneath the upper lid they create a feeling of pressure and pain, due to the fact that the foreign body scrapes the cornea with each movement of the lid; the epithelium may be injured in this way, but even without such injury the terminal filaments of the trigeminus in the cornea are irritated.

The favorite situation of these foreign bodies is the sulcus subtarsalis, which runs parallel with the margin of the lid. When the upper lid is everted the foreign body may be seen and removed with a bit of wet cotton. After it has been removed, the feeling that it is present persists and the patient may solemnly aver that it is still in his eye even when it is on the cotton. In such cases the pain will disappear if a drop of cocain is instilled into the eye. Although the presence of a foreign body is usually betrayed by the pain, yet it may become encapsulated without causing irritation; this is particularly true of grains of powder. A foreign body may, under certain circumstances, lie quietly in the upper transition fold for a long time and then cause symptoms of a chronic conjunctivitis to appear. Sometimes foreign bodies are introduced intentionally into the conjunctival sac. The bad custom prevails in many places of introducing the so called crab's eyes, flat concrements from the stomach of the crab, beneath the upper lid in the hope that by its means another smaller foreign body will be removed. It is not an uncommon occurrence that the crab's eye is discovered months later in the transition fold enveloped in granulations.

Foreign bodies are also introduced into the eye for the purpose of deception. Persons to be examined in regard to an accident sometimes induce an irritation of the formerly injured eye for which there is no natural reason. In one case a patient brought with him a little package of fine, white marble dust, some of which he frequently introduced into his eye in order to simulate a chronic condition of irritation.

The injury produced by a beard of grain forms a typical clinical picture, which is induced by a prolonged continuance of a foreign body in the conjunctival sac. It is not uncommon for a beard of grain to enter the conjunctival sac, to bury its fine sharp ends in the tissues of the upper transition fold, and gradually to excite a severe irritation of the eye, associated with a swelling of the lid and a marked purulent secretion. At first glance the picture resembles that of a severe infectious conjunctivitis and frequently patients state that they consulted a doctor who found nothing in the eye. The expert usually sees at once from the superficial erosion of the cornea that a foreign body must still be present, and after he has cocainized the eye thoroughly he carefully investigates the upper transition fold and finds the bit of beard enveloped in mucus. After the lid has been everted the lower margin of the tarsus is easily lifted up with a glass rod.

The condition presented by our next case is altogether different. This man is a tinsmith. Something flew into his eye two days ago. The eye has a slight ciliary injection and on the cornea may be seen a little foreign body surrounded by a brown ring. This is a particle of iron, or rather of ferric oxide, which was thrown off as a spark and became oxidized. The brown ring consists of hydrated oxide of iron. In other cases the foreign body may be a cinder, or a bit of stone.

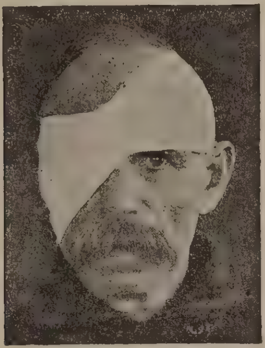

FIG. 67.—Monocular Bandage.

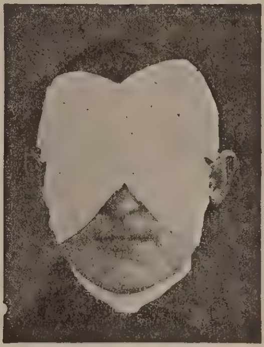

FIG. 68.—Binocular Bandage.

These injuries of the cornea by foreign bodies are the most common of all injuries to the eye; one set of statistics shows that they form from 40 to 60%. In many places they are of daily occurrence, especially among workers in iron and steel. According to *Cohn*, 95% of the workers in a metal industry had metal filings fly into their eyes weekly or even daily; most of these foreign bodies either escaped themselves, or were removed by the fellow workmen, assistants who are not always satisfactory. Some of these workmen calculated that they had suffered from 2,000 such injuries in the course of twenty years.

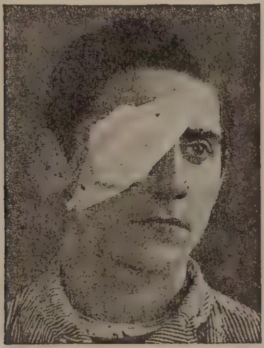

FIG. 69.—Adhesive Plaster Dressing.

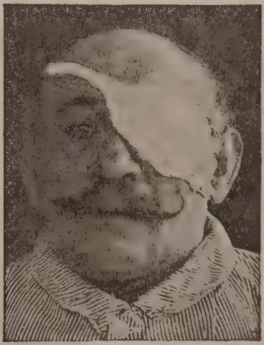

FIG. 70.—Patch.

The eyes of those who work with the grindstones are the most endangered. Among quarrymen both bits of stone and sparks of steel cause injury. The trade of such a man can occasionally be recognized from his corneæ.

A few drops of a 2 or 4% solution of cocain are instilled, and then the foreign body is lifted from its bed with a sterile, platinum needle and the brown edge formed by the particle of iron is scraped away. The surrounding epithelium must be spared as much as possible while this is being done. After the foreign body has been removed some sublimate vaseline is applied to the eye, which is then bandaged, for experience has taught us that the injured epithelium reforms most quickly to cover the wound when the eye is kept as quiet as possible. In addition to this we should do what we can to prevent an infection of the wound. If the foreign body is not removed it may not cause any reaction for several days, but then an inflammatory infiltration appears in its vicinity, the foreign body becomes surrounded by a gray edge of pus cells and is finally cast off from the cornea by this suppuration, leaving an ulcer. Powder grains may remain permanently in the cornea without causing reaction.

The condition is worse when the foreign body is situated in the deep layers of the cornea, or when its point protrudes into the anterior chamber. If it is a splinter of iron the magnet may be used. If this fails, or if the foreign body is not composed of iron it may be necessary to incise the cornea and extract it with forceps. The method of dealing with intraocular splinters of iron will be shown later.

II. Nonperforating Wounds and Erosions of the Cornea

The eye of this mother has been scratched by the finger nail of her baby. The eye is extremely irritated, lacrymates, is photophobic and has a ciliary injection. In the centre of the cornea is a superficial abrasion of the epithelium, which we call an erosion. The extent of the erosion can be determined readily by the instillation of fluorescin. These erosions are often caused by twigs that scrape the surface of the cornea. They are painful and must be treated carefully for two reasons. Among the laboring classes infections are to be feared that give rise to ulcus serpens, and therefore the eye should be bandaged for several days, after sublimate vaseline has been applied, in order that the epithelium may cover the defect from its margin.

In the second place it happens not infrequently that several weeks, months, and even years after the erosion has apparently healed pain and sharp signs of irritation return with an erosion in exactly the same place, although no new injury has occurred; these are cases of recurrent erosion. The recurrence is due to the fact that after the first erosion the epithelium did not become attached with normal firmness to *Bowman's* membrane, so that slight causes are able to elevate it in the form of a bleb which soon ruptures. This is apt to happen in the morning on awaking, when the corneal epithelium is somewhat attached to the conjunctiva palpebræ and is torn away. Such recurrences may be repeated many times. A large quantity of boric acid ointment should be introduced regularly into the conjunctival sac every evening. If the case is obstinate, or if the erosion is large, the diseased epithelium may be scraped off (abrasio corneæ) in order to stimulate regeneration. An erosion is a quite superficial wound of the cornea. Deeper wounds may be lacerated or incised, and their examination is likewise facilitated by the use of fluorescin. The margins of the wound usually swell very quickly, even when there is no perforation, because the fluid of the conjunctival sac is imbibed.

III. Burns

Another superficial injury is exhibited by this patient who has some lime in his eye. The eye is greatly irritated; a large part of the cornea is dull and cloudy, while in the conjunctiva tarsi of the lower lid and in the transition fold are white, scurfy places elevated above the rest of the reddened and swollen conjunctiva. The lime that enters the eye is slaked, or in the form of mortar. A serious inflammation may be excited by manure containing lime. Children place lime in a bottle, pour in water and then the lime and bits of glass from the explosion enter their eyes. Burns are also produced by caustic alkalies and acids, hot water, hot fat, molten metals and hot instruments. Burns with curling tongs are quite frequent among women.

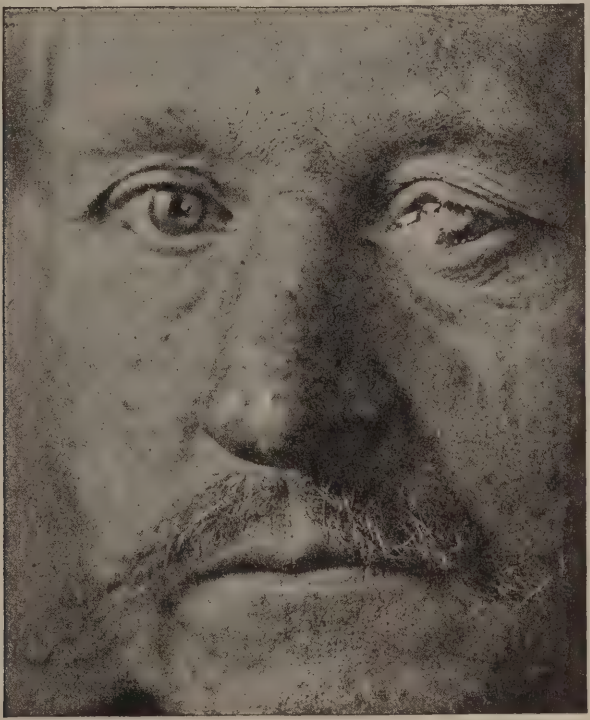

FIG. 71.—Symblepharon after Burn with Molten Metal.

In a fresh case of lime burn all the larger particles of lime are to be removed from the conjunctival sac, the eye washed out with salt solution, and then dressed with ointment. Cocain ointment may be used to relieve the pain. It is of great importance at first to introduce fresh boric acid ointment every hour when possible.

The fate of the cornea is of primary importance. The intensity of the opacity produced in it depends on the depth to which the tissue is destroyed. When the epithelium alone is injured, as in the frequent burns with curling irons, it regenerates in a short time, but after a severe lime burn the cornea may be porcelain white, dry and insensitive. The burned necrotic parts of the cornea and conjunctiva are thrown off gradually and the eye maintains a condition of severe irritation for a long time. Healing of the surface of the wound and the replacement of the loss of substance must naturally take place through the formation of connective tissue.

The result of a burn therefore depends on the situation and extent of cicatrization. The prognosis is very good in slight burns, like those with curling irons, restitutio ad integrum follows, the cornea again becomes perfectly transparent and the slight defect in the conjunctiva heals smoothly.

Deeper burns of the cornea leave opacities that impair the vision. Repeated attempts have been made to clear up opacities of the cornea caused by lime by means of chemicals.

Further serious injury to the eye may result from the cicatrization in the conjunctiva that follows burns, as is shown in this case in which, in addition to the corneal opacity, peculiar, tense adhesions extend between the conjunctiva of the lids and that of the eyeball, when the eye is turned up or down, and prevent the lids from being drawn away completely from the eyeball. The patient was burned long ago with molten metal. These bands are partly fleshy, partly tendinous.

In such a case as this the first thing to be ascertained is whether the symblepharon is complete or partial. In complete symblepharon, which is in fact rare, the lid is adherent everywhere to the eyeball and the conjunctival sac obliterated throughout its whole extent. In such cases the margins of the eyelids are also adherent, as a rule, a condition known as ankyloblepharon. In such grave cases, in which the cornea is usually destroyed, the eyeball can scarcely be seen and a sort of traumatic cryptophthalmos is formed. In partial symblepharon certain areas in the conjunctiva of the lids and of the eyeball which lie in apposition with each other are adherent.

Partial symblepharon is divided into symblepharon anterius and posterius for practical therapeutic reasons. If the adhesion involves the transition fold it is called a symblepharon posterius, but when it forms a bridgelike, cicatricial band between the lid and the eyeball, beneath which a probe can be passed along the transition fold, it is a symblepharon anterius, or bridge symblepharon. Improvement can be obtained by operation only in partial symblepharon.

85. Contusions of the Eye with Rupture of Its External Capsule

Contusions of the eye are divided into two groups, as they are, or are not, associated with rupture of the capsule of the globe. The former injuries are the more serious.

Contusion may produce rupture of the eyeball directly or indirectly. By direct ruptures are meant such as take place at the point where the contusion was received. Such an injury can be differentiated with difficulty from a lacerated wound of the eye and no sharp line can be drawn between them, but it may perhaps be well to remember that direct lacerations occur more often in the cornea than in the sclera. Usually a curved, flaplike wound is produced associated with grave complications within the eye.

Indirect ruptures of the eye, which are much more common, occur at points at some distance from the place struck by the object that produced the injury. To this group belongs this typical case of indirect rupture of the sclera with traumatic aniridia, hæmophthalmos and luxation of the lens beneath the conjunctiva. The woman states that she was struck from below with a piece of wood four days ago. We see a large hemorrhage beneath a considerable area of the conjunctiva scleræ; the cornea is intact and clear; the anterior chamber is seen by focal illumination to be abnormally deep, while at its bottom is a fresh hyphæma. The most striking symptoms, however, are two: the iris is looked for in vain, it is totally absent. When the upper lid is raised a dark gray line may be seen in the sclera some millimetres from the margin of the cornea, which begins above and runs concentrically rather obliquely inward for more than a centimetre. At one place it is covered by a round body which is covered everywhere by the untorn, intact conjunctiva. This is a rupture of the sclera with the lens, that has been extruded from the eye, lying upon it beneath the conjunctiva. Dark brown masses may be seen close to the lens and here, evidently, is the iris, torn with the lens out of the eye and now lying beneath it and caught in the margins of the wound in the sclera. At the present time the intraocular tension is the same as that of the uninjured eye, whence we conclude that the margins of the tear in the sclera are already luted together, for otherwise intraocular fluid would continue to escape beneath the conjunctiva. In some cases, especially when the conjunctiva also has been torn, the tension will show that a great part of the contents of the eye, especially the vitreous, has escaped, a circumstance that renders the prognosis worse. In our case the injury has performed an interesting experiment: the entire iris has been torn out and still the eye has regained its tension after closure of the wound. Hence the intraocular fluid, particularly the aqueous, can be reproduced without the iris, one of the evidences we have that the ciliary body is the principal source of the aqueous. But our patient does not come to us because she is interested in this experiment, she wishes to know what will become of her eye. A dark red reflex is obtained from the vitreous by focal illumination; a view of the interior of the eye cannot be obtained with the ophthalmoscope; vision is reduced to the perception of light and darkness; the projection is uncertain. Hence we formulate the diagnosis: Subconjunctival indirect rupture of the sclera with traumatic aniridia, luxation of the lens beneath the conjunctiva and hæmophthalmos. The usual site of the scleral rupture in such a case is parallel to the corneal margin, either directly above or above and inward.

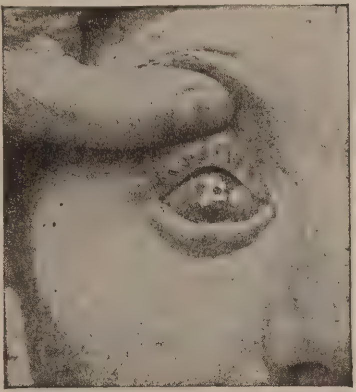

FIG. 72.—Rupture of the Sclera with Subconjunctival Luxation of the Lens.

When we analyze this clinical picture the first question suggested is: How is this characteristic indirect rupture of the eyeball brought about with its peculiar predilection for the corneoscleral margin? Many theories have been advanced to solve this interesting mechanical problem, but without going into their details we may note the following points. The eyeball is usually driven against the wall of the orbit by the blow; the compression causes a considerable increase of the intraocular tension; the capsule of the eyeball is thereby overstretched and rupture takes place at the point of least resistance. This place is the corneoscleral margin for anatomical reasons. According to *Mueller* the rupture takes place in a meridian intermediate between the point of attack and the point of resistance, usually in the region of *Schlemm's* canal. The ligamentum pectinatum, which forms the posterior wall of *Schlemm's* canal, yields first and the rupture follows from within outward; first the sclera is torn, then the episclera and then perhaps the conjunctiva. If the rupture extends farther along the surface it follows by preference the line along which the anterior ciliary vessels perforate the sclera, where it is evident that the strength of the tissue must be less, and extends several millimetres back from the margin of the cornea in correspondence with the course of these vessels. The site of the rupture at that place is very distinct in our case.

Sometimes small ruptures of the globe are met with which are situated above and internally to the margin of the cornea. In such cases the laceration passes directly through the coats of the eye and is close to the limbus.

In accidents the rupture of the eyeball is the quick result of an unfortunate turn of the eye. Experiments, in which the direction and the force of the blow can be changed as desired, have been made to clear up the origin and mechanics of ruptures of the eyeball. In those made by *Mueller* the ruptures usually took place in a meridian of tension. The other symptoms, hemorrhage and laceration of tissue, need no explanation.

Very often the conjunctiva is torn in these ruptures; this can be recognized immediately. When the eyeball is shattered in this manner we meet with such complications as prolapse and tearing away of the iris, luxation of the lens, extensive loss of vitreous and great intraocular hemorrhages.

When a case of subconjunctival rupture of the globe enters his office it is the duty of the physician to make accurate notes of the condition present, that later he may be able to testify correctly concerning the condition of the eye after the injury. Then he should apply an aseptic protective dressing and send the patient to the nearest ophthalmic hospital, where our duty begins. In this case two therapeutic indications are to be met. We have learned that the rupture has closed and that the intraocular tension is again normal, but the configuration of the eye has been changed by the pressure of the luxated lens beneath the conjunctiva upon its surface. As soon as the closure of the wound is firm enough the lens, which is now to be regarded as a foreign body, is to be dissected out, and the wound made in the conjunctiva for that purpose closed again. We must endeavor to preserve the form of the eyeball so far as possible and to induce resorption of the large hemorrhage in the vitreous.

Unfortunately the hemorrhage in the vitreous can be influenced very little therapeutically. The proposal I made to bring about solution of the blood by the intraocular injection of specific hæmolytic serum, was frustrated by the fact that foreign kinds of serum are not borne well and we can scarcely inject sufficient complement with the immune bodies. Hence nothing now remains but to try sweating, potassium iodide and subconjunctival injections of salt, at least for a while, in order to stimulate the resorption of the hemorrhage. We meet with the most wonderful things in the course of these contusions. In grave cases the hemorrhage finally organizes, the vitreous shrinks and atrophia bulbi is the result; but lucky cases are also met with. These clinical pictures are of particular frequency among farmers as the result of being gored with a cow's horn, and it may happen that the lens may thus be extruded from the eye, which becomes aphakic, as after an operation for cataract, and good vision preserved. I have indeed seen one patient in whom an extraction was performed in this way by a cow, first on one eye and a year later on the other, with good subsequent vision, but, as a rule, the prognosis of such grave contusions is not good, and many eyes thus injured have to be enucleated.

Some deviations from the typical indirect rupture of the sclera are met with. The length of the tear may vary; on the average it is from 6 to 12 mm long, but it may be smaller.

The patients are usually elderly, because the rigidity of the sclera favors the occurrence of such lacerations, and their occupation is of great importance, yet indirect ruptures of the sclera are not so very rare among children. It has been observed that an eye may suffer from such an injury twice and yet a comparatively good result be obtained.

The lens is usually extruded from the eye, or it may be caught between the margins of the wound. When luxated beneath the conjunctiva it usually covers the place of rupture, but it may be dislocated far toward the transition fold beneath the conjunctiva.

Exceptionally, the rupture of the sclera may lie not at the corneoscleral margin, but at the equator, or in the posterior segment of the globe, and then the lens escapes into *Tenon's* space. The lens is usually luxated in its capsule in these cases, but the capsule may be torn and remain in the eye after the escape of the lens. I have performed a successful discission in one such case. The luxation is usually associated with at least a partial avulsion of the iris, so that an iridodialysis, or a broad coloboma, is formed. The portions of iris that are left often become inverted.

Although aniridia and luxation of the lens are associated, as a rule, when the iris is totally avulsed, yet cases have been met with in which the iris alone was torn away and extruded beneath the conjunctiva, while the lens remained in its proper place and position.

When it is possible to see into the eye, in the later stages of an indirect rupture of the sclera, the ciliary processes are frequently invisible, although the iris is wholly absent. They may have been flattened and drawn out of sight by an atrophy of the ciliary muscle, or it may happen that they have been drawn inward by adhesions and may be perceived by oblique illumination.

The place of rupture remains visible later as a bluish black line. Even when the scar is smooth such eyes have a great deal of corneal astigmatism, or the eyeball may assume a conical form. The scar may also yield and give rise to a scleral staphyloma, which may lead to increased tension.

A very great pigmentation of the conjunctiva may originate from such a rupture of the sclera, and the displaced pigment of the uvea may spread even to the transition fold.

Finally, the important fact deserves mention that, in such a case of indirect rupture of the sclera with preservation of the conjunctiva intact, there is no danger of sympathetic inflammation of the other eye. In the rare cases in which a sympathetic iridocyclitis has occurred there were without doubt minute lacerations of the conjunctiva, through which the agents of inflammation found their way into the eye.

86. Contusions of the Eye without Rupture of Its Capsule and the Clinical Pictures Produced in the Anterior Segment of the Eyeball

Contusions are injuries of the eye and of its environs produced by a blunt impact in which the foreign body does not penetrate the tissue. When the eyeball itself is struck the contusion is direct; when the concussion falls first on the environs, or more distant parts, and is transmitted to the eyeball, the contusion is indirect.

The common feature of all contusions is that they produce mechanical changes in the affected tissues. These changes consist of compression, displacement of the tissue elements against each other, separation, laceration and displacement of entire parts with relation to each other, and the most manifold clinical pictures are produced by the combinations of these mechanical effects.

The consequences of a contusion, especially of the eye, depend on several factors: 1, the nature, form and size of the object that produced the injury; 2, the force and velocity with which the eye is struck; 3, the direction from which the blow comes and the situation of the point where it is received. In general the process is as follows:

Apparently the eye presents a simple, elastic, nearly spherical capsule filled with fluid, yet this is not the case. The cornea has a shorter diameter than the sclera and therefore there is a flat surface at the limbus; the coats that form the capsule have little expansibility; its contents are by no means uniformly fluid. In front is the aqueous, behind the jellylike vitreous. The interior of the eye is also not a single cavity, for the lens and zonule separate the anterior fluid contents from the vitreous, and the iris is stretched between the anterior and posterior chambers, leaving the pupil as the only communication between them.

When an eye suffers a contusion the place struck by the foreign body is pressed in and bruised. Neighboring parts must yield, the capsule of the eye is stretched and may even be torn on its inner surface. Thus we meet with lacerations of the inner surface of the cornea (infractio corneæ). When the sclera is severely indented the chorioid and retina may be torn. Direct rupture of the chorioid and retina is rare.

In many cases the local changes at the place where the blow is received are slight. The pressure is distributed uniformly, by the fluid nature of the contents of the eye, in all directions upon its contents, as well as upon its capsule. This increase of tension causes the capsule to be stretched, the eye is made to assume more nearly a spherical form, and the displacement of the aqueous and vitreous by the local flattening at the place struck affects more distant places and contributes to their distention. Thus it happens that in a contusion portions of the eye far removed from the place of impact may indirectly suffer severe injury.

This transmission of pressure is not all. The eye is displaced at the moment of injury in a manner that varies with the direction of the blow. If, for example, the eye is struck from in front in a sagittal direction it must be driven backward into the orbital fat. The force is then expended on this cushion, and thus the eyeball is protected under certain circumstances. It has been claimed, though not without dissent, that the optic nerve may be driven in this manner directly into the eye.

If the direction of the blow is not through its centre the eyeball must be driven to one side and, when the direction is tangential, it may be rotated. From this it is evident that the mechanics involved in a contusion of the eye may be very complicated, and this is in harmony with the fact that the clinical pictures met with are manifold.

The complicated anatomical structure of the eye also needs to be considered. The membranes of the eye vary in structure, elasticity and power of resistance. The eye contains muscles which may be spasmodically contracted; vessels and nerves pass through the membranes. These conditions are such that a more accurate mechanical analysis cannot always be made of the individual morbid changes. The number of theories that have been advanced is almost proportional to the number of scientific publications.

The first question to be answered in the examination of a contusion of the eye is whether the capsule is, or is not, ruptured. Contusions of the eye are divided into these two groups for an important reason. Both groups frequently have such symptoms in common as hemorrhage and displacement of tissue, but they almost always differ in one point. *Contusion with rupture of the capsule of the eye is always the more serious.* Thus an important point in the prognosis of the injury is obtained at a glance.

Rupture of the capsule is the easiest to recognize of all the changes met with in contusion of the eyeball; its favorite seat is near and concentric with the corneoscleral margin.

Nothing of this sort is to be found in the patients here present, and both patient and physician breathe more freely, for in contusions without rupture the prognosis for preservation of the form of the eyeball is better. Now our duty to analyze the individual injuries commences, for the clinical pictures produced are very manifold. In order that no real symptoms may be overlooked and to indicate what is worthy of note in such a case I have arranged in the following table the most important pathological changes that occur in contusion without rupture of the eyeball.

Tabulated Abstract of the Clinical Pictures Met with after Contusions of the Eye without Rupture of the Outer Capsule.

1. Hemorrhages into the eyelids.
2. Hemorrhages beneath the conjunctiva.
3. Cloudiness and erosions of the epithelium of the cornea.
4. Hyphæma.
5. Iridodialysis and irideremia.
6. Radiating lacerations of the iris.
7. Traumatic mydriasis and meiosis.
8. Laceration of the sphincter and of the individual layers of the iris.
9. Inversion of the iris.
10. Paralysis and spasm of the accommodation.
11. Cataract.
12. Subluxation and luxation of the lens.
13. Hypotony and hypertony.
14. Hemorrhage into the vitreous.
15. Commotio retinæ.
16. Retinal hemorrhages. Changes in the macula.
17. Detachment of the retina.
18. Chorioidal hemorrhages with patches of pigmentation and decolorization.
19. Rupture of the chorioid.
20. Crushing and laceration of the optic nerve; hemorrhages into its sheath.

With this table before us we will now examine our patients. It is self evident that every possible combination cannot be presented.

This case presents the usual picture of a contusion of the eye and some of the changes to be observed in its anterior segment can be detected at once. There is a marked swelling and an *ecchymosis of the lids.* Our first concern is to see that the eyeball is intact, so we cautiously lift the upper lid with the elevator in order to bring the globe into view. There is no rupture anywhere, but there is an extensive subconjunctival hemorrhage which shows that the injury caused lacerations of the episcleral vessels from which blood has poured forth beneath the conjunctiva bulbi. When the escape of blood is slight the place where the eyeball was struck can frequently be recognized from the hemorrhages, which are sometimes striated. We have also to notice on the cornea whether there are any little erosions of the epithelium.

In this patient the most striking symptom is a red band several millimetres high at the bottom of the anterior chamber, a hemorrhage into the anterior chamber that is called a *hyphæma.* The horizontal demarcation is characteristic. As long as the blood remains not coagulated it readily changes its place in different positions of the head. As a rule such a hemorrhage comes from lacerations of the vessels of the iris and sometimes the place from which the blood trickles can be seen. At other times its origin cannot be determined. It may come from a laceration of *Schlemm's* canal.

When such a case as this enters the office, and presents all the signs of a contusion with changes in the *anterior* segment of the globe, it is necessary to examine the eye with the ophthalmoscope and to test the vision in order to guard against trouble later. As we shall see in another case grave changes may be present in the posterior segment also.

No treatment is necessary in this case other than a hot, wet dressing. When erosions of the epithelium are present cocain ointment or boric acid ointment may be used. As a rule the hyphæma is absorbed in a few days. The swelling of the lids persists for some time and the well known changes of color take place during the resorption of the hemorrhages in the skin and conjunctiva.

The second patient received a blow from a fist on his eye some time ago. The most marked symptom is a change in the shape of the pupil, which is no longer round, in that its lower margin forms a straight line. At the ciliary margin of the iris below may be seen a little black segment where the iris has been torn from its attachment and we may look into the eye with the ophthalmoscope through this aperture in the same way as through the pupil when the lens is not opaque.

When the *iridodialysis* is of greater extent the ciliary processes, the margin of the lens and the fibres of the zonula become visible.

The manner in which an iridodialysis is produced was explained by *v. Arlt* in this way. When a blunt object strikes the cornea the latter is flattened. As the fluid in the eye is not compressible the flattening of the cornea must occasion a change in the shape of the eye consisting of a sudden dilatation of the corneoscleral ring and the iris is thereby torn loose from its insertion. Another view is that the contraction of the pupil at the moment of injury plays a part in the origin of iridodialysis. We learn from experiments that a marked meiosis may follow contusions of the living eye. *Mueller* believes that the foreign body presses in the sclera at the limbus, that the ring of insertion of the iris is thereby partially stretched and the sudden contraction of the sphincter and dilatator brings about the avulsion of the iris.

The iris does not usually become reattached to its insertion; when the ciliary insertion has once been torn and the margin of the pupil has been changed to a straight line by the shortening of the sphincter, the separation is permanent.

Therapeutically we can do nothing. This is not necessarily a misfortune as vision is not impaired in most cases of iridodialysis, especially when the tears are small. When the opening is quite large monocular diplopia may be occasioned, because an image of an object may be thrown on the retina through both the pupil and the peripheral aperture in the iris. In such cases a suitable stenopæic lens is of service.

The size of an iridodialysis may vary a great deal. Sometimes it is so small that it can scarcely be detected, in other cases the entire iris may be avulsed from its ciliary insertion; when the latter happens the iris is found lying at the bottom of the anterior chamber folded into a little coil, where it undergoes further shrinkage.

Here is another form of laceration of the iris after contusion without rupture. At first glance the pupil of this patient's eye seems to be moderately dilated, but a more careful examination shows that its margin is quite regular above and to the inner side and bordered by a dark edge of retinal pigment, while below and to the outer side this edge is more or less wanting and there are little notches in the margin. The pupil appears to be slightly dilated because the margin has retracted somewhat at this place.

These radiating lacerations extend to the ciliary margin of the iris in very rare cases. The margins of such a tear gape widely and lack the edge of retinal pigment, while the pupil assumes a colobomalike, or pointed arch form. The diagnosis in our case is that in consequence of the contusion *lacerations have taken place in the sphincter pupillæ.* This clinical picture is called *traumatic mydriasis.* Such tears in its margin form the most common cause of the dilatation of the pupil observed after contusions.

Various theories are maintained concerning the way in which these lacerations are brought about. According to *Franke* the force of the stretching of the iris in the spasmodic contraction of the pupil and the dilatation of the corneoscleral ring is expended first upon the rigid ring of the sphincter and produces laceration. Other writers, like *Foerster*, ascribe the lacerations to the effect produced by the backward driven aqueous. The pupillary part of the iris is pressed firmly against the lens, while the marginal portion that extends beyond the lens is pouched out toward the posterior chamber. This saclike protrusion of the periphery of the iris and the sudden dilatation of the pupil cause manifold changes, overstretching of the muscles of the sphincter, and lacerations.

There is no treatment. Usually a slight dilatation of the pupil persists and may compromise vision somewhat. If the sphincter is simply overstretched and not torn the mydriasis may pass away. In fresh cases of such contusions a bandage should be worn for several days.

Cases are met with after a contusion in which part of the iris is bent backward, so as to give the impression that an iridectomy has been performed. The folded back iris lies on the ciliary body where it is usually made fast by hemorrhages and exudates. Such an *inversion* of the iris occurs even with simple contusions, but more frequently in connection with ruptures of the sclera.

The opacities produced in the cornea by contusions are variable. The surface may be dull and dotted, the epithelium may show erosions. In this case, in which the eye was struck by a whiplash, there is a deep, cloudy opacity, which is supposed to be an œdema with swelling of the cornea, due to injury of the epithelium of the posterior surface.

Doubtless *lesions of the endothelium and lacerations of Descemet's membrane* take part in the deep, striated opacities of the cornea that follow bruises of the eye produced by the application of forceps. Their presence has been demonstrated anatomically many times when the eyes have been injured during labor. *Stock* has shown that a keratoconus may develop in such cases. These lacerations of *Descemet's* membrane are at a distance from the place of impact and are therefore indirect. Direct partial ruptures of the posterior layers of the cornea also take place when the cornea has been violently pressed in.

When hemorrhages are present in the anterior chamber it must be borne in mind that they may come from an isolated rupture of *Schlemm's* canal. Hemorrhages in the anterior chamber of an otherwise healthy eye are absorbed with comparative quickness. They frequently give a greenish yellow discoloration to a light colored iris.

When the anterior chamber is filled with blood and the resorption is slow the clinical picture may develop that we call *imbibition of blood by the cornea.* A disclike opacity forms in the central part of the cornea while the margin remains free. The coloring matter of the blood enters by diffusion from the anterior chamber and here undergoes further changes. When we examine the cornea anatomically we find that it is diffusely permeated with ferruginous blood pigment and in addition observe in the opaque area numberless little, strongly refractive granules which were thought by *Leber* to be fibrin, by *Vossius* to be fibrillæ of the cornea that had undergone hyaline degeneration. I think it probable that they come from the albuminoids of the hæmoglobin. It is characteristic of this imbibition of blood by the cornea that the disclike opacity disappears gradually in the course of years.

In other cases of severe contusion of the eye a gelatinous transudate, rich in albumin and containing fibrin, appears in the anterior chamber and renders the aqueous very opaque. This transudate comes from the paralyzed vessels of the iris and ciliary body.

Whenever we meet with an iridodialysis we may be sure that the eye has suffered a severe contusion. It should be mentioned, as a supplement to my former statement, that an iridodialysis has been observed to heal spontaneously in rare cases. When the lens has been partially injured a patient may use the aperture formed by the iridodialysis for visual purposes. Complete avulsion of the iris without rupture of the eyeball is rare; laceration of the sphincter is much more common. The dilatation of the pupil and the slowness of its reaction are due not alone to the notching, but also to a paralysis of the sphincter.

In rarer cases only the posterior pigment layer of the iris is torn. By focal illumination the red of the fundus may then be seen to shine through the iris, or, when cataract is simultaneously produced, the opaque lens may be seen through this aperture.

When we find an inversion of the iris that simulates a coloboma we may be sure that the lens is subluxated, for the zonule is torn in conjunction with the inversion of the iris. This retroflexion of the iris can be distinguished from a coloboma by the facts that the corners of the coloboma are absent and the ciliary processes cannot be seen. Finally, disturbances of the movements of the pupil are very commonly produced by contusions even without lacerations of the sphincter. We meet with a traumatic mydriasis and meiosis. In traumatic mydriasis the pupil often is irregularly dilated, oval, or partially pouched out, and the reactions to light and convergence are very slow. Traumatic mydriasis is due to injury of the sphincter and of its nerve fibres, caused by increased pressure, distortion of the tissues, or an overstretching of the muscle fibres (*Foerster*). A traumatic meiosis dilates very badly under the influence of atropin. A spasm of the sphincter is present which is favored by a serous saturation of the iris. While traumatic meiosis usually disappears in a few days mydriasis generally persists and often furnishes the only evidence of a former contusion.

87. The Clinical Pictures That Appear in the Posterior Segment of the Eyeball after Contusions without Rupture of the Sclera

This boy has a contusion from having been struck in the eye with a snowball. The lid is somewhat swollen and ecchymosed, and there is a little wound in the skin at the upper margin of the orbit. He can open his eye somewhat, but complains that he can see very little with it.

Our first care is to ascertain whether the eye has been perforated at any place, that is whether the contusion is or is not associated with rupture, for the presence of a rupture makes the prognosis of such an injury very grave. No rupture of the sclera is to be found in this case, and no changes are to be seen in the anterior segment of the globe, except some little subconjunctival hemorrhages, and these are insufficient to explain why the eye can no longer see. Learn from this case that in contusions the physician must not limit his examination to the anterior segment of the globe. Even when changes are present there, complications may exist within the eye, and when no changes are present in the anterior segment an intraocular complication must be the cause of the disturbance of vision, so the first question is: Has the vitreous suffered from the contusion?

1. Changes in the Vitreous after Contusions of the Eye

The most important changes in the vitreous that follow contusions are hemorrhages which vary in form and in size. The blood may come from either the ciliary body, the chorioid, or the retina. Circumscribed hemorrhages move about here and there as flaky, striated masses. In graver injuries the vitreous may be diffusely infiltrated with blood, so that the fundus cannot be seen and only a red reflex can be obtained by focal illumination. Vision may be reduced to such a degree that perception of light and projection are hardly present. We cannot at this point exclude detachment of the retina.

The resorption of hemorrhages into the vitreous is very slow. At a later period grave injuries may be discovered in the fundus, such as lesions in the chorioid and retina, with proliferation of pigment, atrophy of these membranes, detachment of the retina and atrophy of the entire eyeball.

An eye with hemorrhage into the vitreous after contusion should be treated by a bandage, subconjunctival injections of salt and the internal administration of resorbents. But such a hemorrhage is not the cause of the disturbance of vision in this case.

2. Changes in the Retina in Contusions without Rupture of the Globe

Hemorrhages may be present in the retina even when they are not to be found in the vitreous.

Laceration of the retinal vessels is frequently preceded in experiments on animals by a high degree of ischæmia which disappears in an extremely short time and therefore is scarcely to be observed in man. As long as the hemorrhages are small they lie in the substance of the retina itself, but they may occur between the retina and the vitreous.

In these so called *preretinal hemorrhages* it is not necessary that the eyeball itself should have been struck. Occasionally they are observed after grave injuries to the head and are caused by the engorgement due to the serious intracranial disturbance of the circulation. The preretinal hemorrhage appears ophthalmoscopically as a large, round, dark red disc. If it exists long enough for the blood to have time to sink, its upper edge finally becomes horizontal.

The formation of aneurysms of the retinal vessels has been reported in rare cases of contusion of the globe. It is self evident that hemorrhages in the retina may take part in the reduction of vision after contusion, especially when they are situated in the region of the macula.

Retinal hemorrhages are generally resorbed more quickly than hemorrhages into the vitreous, but they may produce further changes in the fundus, such as decolorized spots and patches of proliferated pigment in its innermost layers, and may also give rise to connective tissue bands that produce the picture of *retinitis proliferans.*

In this case there are no retinal hemorrhages, but a large part of the retina appears opaque and gray white, the opacity extending far toward the periphery. The retinal vessels stand out very distinctly by contrast in this superficial, gray white opacity, but exhibit no tortuosity, which differentiates the condition from detachment of the retina; neither can any undulations of the membrane be seen.

This is a typical case of *retinal œdema after contusion.* This opacity was described first by *Berlin* and has therefore been called *Berlin's* opacity. A severe contusion, in this case by a snowball, is necessary for its production. The white decoloration of the retina reaches its maximum, as a rule, in twenty-four hours, and then passes away in the course of three or four days, the clearing up beginning at the periphery with special reference to the retinal vessels. In rare cases only, a permanent decolorization and pigmentation of the fundus is left. According to *Haab* a special opacity of the macula may occur together with this superficial, peripheral opacity of the retina.

When we consider the severity of the contusion it is conceivable that other complications may be present, at least a ciliary injection, and corneal changes, traumatic mydriasis, hemorrhages into the anterior chamber, hypotony and even emphysema of the orbit have been observed.

Many experiments have been made to determine the origin and nature of this contusion opacity. It is an œdema of the retina, brought about by transudation in consequence of the traumatic vascular paralysis, especially of the chorioidal vessels. The fluid enters the retina by way of diffusion. The retina and chorioid suffer laceration and distortion by the stretching of the capsule of the eyeball and it is certainly conceivable that the retinal elements may be harmed by the œdematous saturation. At any rate the cause of the visual disturbance lies primarily in the retina itself, although other complications must be taken into account. The field of vision may be contracted and disturbance of the light sense may be present, but usually the patients are troubled by a reduction of the central vision, and this visual disturbance may last a long time after the involution of the opacity. The greatly increased tension caused by the contusion may bring about a direct pressure paralysis of the nervous elements of the retina. The macula lutea itself is very often injured in contusions.

Yellow spots, pigmentation and even a total atrophy may remain after a *contusion opacity of the region of the macula.* A short time ago we had a patient to investigate who had been mistaken for a malingerer. Such a change in the macula following a contusion had been overlooked. He had a distinct central scotoma with greatly impaired vision.

Hemorrhages situated in the macula take part in the disturbance of vision, and masses of connective tissue and pigmentation may develop from them.

Thirdly, peculiar *holes* occur in the macula after contusions. A round, red disc of about one-half the diameter of the papilla is found at the macula, where the chorioid lies uncovered, or with only slight traces of retinal tissue. It is characteristic of these holes in the macula that changes of the pigment epithelium, decolorization and pigmentation are absent. The temporal half of the papilla may become pale as a sign of the destruction of the papillomacular optic nerve fibres, as *Haab* has shown. These holes in the macula are produced either by a mechanical laceration (*Haab*), or by a softened condition of the macula (*Kuhnt*).

Occasionally the retina is torn as the result of a contusion. The margins of the rupture may turn over and roll in and result in an extensive detachment, or in connective tissue cicatrization, to which the hemorrhages that are usually present contribute not a little.

The connection between contusions and *detachment of the retina* is of more importance to the physician, for we are called upon very often to decide whether a retinal detachment can be ascribed to such an accident, and in such cases we need to differentiate whether the detachment is primary or secondary. After a severe contusion a hemorrhage from the chorioid may push off and detach the retina. Circumscribed detachments are to be found, especially in the region of the macula, after hemorrhages produced by laceration of the chorioid. When the refractive media permit an examination we find in a hemorrhagic detachment a hemispherical prominence covered by a tense retina without folds and a gray red color of the detached portion. A primary detachment of the retina may also be produced after a contusion by the escape of a serous transudate from the paralyzed vessels of the chorioid. The detachment must be considered secondary when the results of the contusion were slight and the detachment appeared at a later time; in these cases hemorrhages into the vitreous, ending in contraction and cicatrization in the chorioid, play a part. Frequently the differentiation, whether the case is one of primary or of secondary detachment, is not easy, and when several years have elapsed since the perfect healing of a contusion the question whether a detachment that appeared later can be ascribed to the accident is always difficult.

The optic nerve itself may be involved in every serious injury. When the injury is fresh we may find hyperæmia, little hemorrhages on the papilla, or a cloudy swelling of the neighboring tissues. Later the optic nerve is frequently pale and atrophic, especially when chorioretinitic decolorizations are present, or the macula has been destroyed.

It is very rare that all the changes to be met with in the posterior segment of the eyeball without rupture of the capsule can be demonstrated in a single case. In this patient the retinal changes form the focus of interest and there are no serious changes in the chorioid.

Finally I have two cases with which to demonstrate some important changes that we meet with in contusions of the eye. The first patient received a blow from a fist on the eye some months ago. Externally the eye is normal. With the ophthalmoscope a yellowish stripe may be seen running concentrically to the papilla on its temporal side. This is a *rupture of the chorioid.* Notice that the retinal vessels pass over it. Ruptures of the chorioid originate in two ways from contusions of the eye. They are either direct, formed at the place of impact, or indirect, at a distance from it.

A direct rupture is much more rare than an indirect. It usually lies so far forward that its anterior end cannot be seen with the ophthalmoscope, and forms a broad, irregular, superficial aperture in the chorioid, through which the white sclera is visible.

Indirect ruptures, like the one in this patient, are more common. It has been calculated that one such rupture is met with in about 1,000 eye patients. Ordinarily they are situated on the temporal side of the papilla, either in the macula, or between it and the papilla, and when they run concentric to the latter they have generally been caused by a blow with a broad surface of impact.

More irregular ruptures are to be seen when the force was applied laterally and with a small area of impact, as for example by a ricocheting shot. Several ruptures may be present; I counted in one case four running parallel to each other. The most striking symptom in a fresh case is the absence of a large hemorrhage, though the retina at this place may appear cloudy and exhibit some hemorrhages. It is supposed that the retraction of the torn vessels prevents bleeding, but in some cases it is evident that a hemorrhage originally covered the site of the rupture. The second clinical peculiarity of the rupture of the chorioid is that usually its color is yellowish white, never the bluish white of the sclera. This is because the entire thickness of the chorioid is not, as a rule, torn through, but the lamina fusca remains. The rupture is permanent. A pigmentation is formed later at its margin. When the rupture is broad ciliary vessels may occasionally be seen in it, looking like red stripes.

The retinal vessels may be seen to pass over the rupture, but it has long been known that the outermost layers of the retina are torn at the same time as the chorioid, and frequently the rupture involves the entire retina. This involvement of the retina, as well as the position of the indirect rupture of the sclera, suffices to explain why the vision is impaired. When the macula is directly affected, as in this case, we find a central scotoma. Other contractions of the visual field have been observed. The injury to the vision is generally permanent. Detachment of the retina has been repeatedly observed to take place in the course of cicatrization and later we often find an extensive retinal pigmentation at some distance from the rupture. Subconjunctival injections of salt may be tried therapeutically when there are simultaneous hemorrhages.

A question of mechanical and physical interest, though a matter of indifference from the point of view of practical medicine, is how such ruptures take place in the chorioid while it is lying between two other membranes. Numerous hypotheses have been advanced to account for this phenomenon. *v. Arlt* thought that the flattening at the place of impact, the dilatation of the equator of the globe, and the stretching of the chorioid produced in this manner was the cause of the rupture. According to *Becker* the posterior segment of the eye is pressed against the optic nerve and the latter is invaginated to a certain extent. According to *Hughes,* rupture of the chorioid is due to stretching and laceration of the eye by the optic nerve. According to *Lohmann,* the rupture takes place at the point where the tension changes to the opposite direction. I believe with *Stoewer* that the real factor is the unequal elasticity of the sclera and chorioid.

Detachment of the chorioid has been observed in rare cases of contusion. This may be caused by a large hemorrhage, or by a subchorioidal œdema. The detachment of the chorioid after cataract operations, which has been described already, is much more frequent. I need hardly add that hemorrhages are prominent among the lesions in the chorioid that follow contusion. These may occur either with or without rupture and may be either in the tissue of the chorioid itself, or in the suprachorioidea. In the latter case the chorioid is pressed up from the subjacent tissue and the picture of a flat detachment of the retina is produced. A chorioidal hemorrhage frequently breaks through the retina and produces a hemorrhage into the vitreous. We do not need to mention again the visual disturbances caused by these hemorrhages.

In all cases in which chorioidal hemorrhages have occurred we must expect that extensive patches will be formed in the fundus, characterized on the one hand by decolorization, on the other by pigmentation. A connective tissue thickening of the chorioid, and adhesions between it and the retina, are formed through the laceration and thrombosis of its vessels; through this the pigment epithelium of the retina has to suffer. The outer layers of the retina also are destroyed, the stroma is thickened and pigment wanders into its tissue. This decolorization and pigmentation of the fundus usually appears months later, only in the rarest cases does a sort of acute pigmentation appear in the fundus immediately after an injury. The beautiful atlas of *Oeller* contains a picture of a case of this nature.

Hypotony and Glaucoma after Contusions of the Eye

The most important clinical symptom in this patient is the increased tension of the eye. The man, 40 years old, was struck in the eye by a rubber tube and the only thing we find, except for some slight lesions in the cornea, is this increased tension. This teaches us that the change of fluids in the eye may be materially influenced by such an injury, and we have now to determine the cause of the glaucoma. Secondary glaucoma caused by luxation of the lens is a frequent symptom after a contusion. It can also be understood that a real attack of glaucoma may be excited by an injury when there is a predisposition to this disease. Other causes of traumatic glaucoma are hemorrhages, either primary or secondary, and gelatinous transudations into the anterior chamber. The signs of contusion are often so slight that the glaucoma seems to be primary.

In this case I can demonstrate something that happens more rarely: By means of *Zeiss'* loupe we may see something that looks like a clear, transparent drop that hangs down from above into the anterior chamber and quivers with every movement of the eye. It is not a subluxated lens; that organ is in its normal place and position; it is a large prolapse of the vitreous into the anterior chamber. A rupture of the zonule must evidently precede the occurrence of this complication, but it cannot be demonstrated as the lens is normally fixed. The vitreous in the anterior chamber must be looked upon as the cause of the increased tension and we shall try to remove it.

We may also meet with the opposite condition, a marked hypotonia, after contusion. In such cases the ciliary body is much irritated, as is shown by the ciliary injection and tenderness, the anterior chamber is deep, the iris somewhat hyperæmic and the tension much lowered. It is plain that in such cases the injury brings about a vascular paralysis in the ciliary body with hemorrhage and œdema.

The irritation of the ciliary body may also manifest itself in a spasm of accommodation, which increases the refraction and produces the so called traumatic myopia. This spasm of accommodation generally passes away spontaneously in a few days, or the traumatic myopia may disappear under the influence of atropin. A traumatic myopia after a contusion may arise in other ways. The zonule may be relaxed as the result of stretching, so that the young lens becomes more convex, or the myopia may be caused by subluxation or luxation of the lens. A myopia due to a lengthening of the axis of the eyeball is unknown as a result of a contusion.

88. Subluxation and Luxation of the Lens after Contusion

The history of this case is that of a contusion.

The eyeball is not ruptured and any injury it may have suffered must be in either its anterior, or posterior segment. No lesions are to be detected in the lids, conjunctiva, or cornea, so we examine the anterior chamber by means of focal illumination. In this patient its upper part is deeper than the lower, and when he is directed to look in various directions the iris is seen to tremble with the movements of the eye. These symptoms prove that the lens is either insecurely fastened, or is dislocated.

The lens is either in an oblique position, one margin directed somewhat forward, the other backward, or it has been displaced laterally, so that it no longer lies in the centre of the fossa patellaris of the vitreous. It may be dislocated to the right, left, upward, or downward. In this case the anterior chamber is shallower below, showing that it is dislocated downward.

The margin of the lens is now to be looked for by means of focal illumination and the ophthalmoscope. We first try focal illumination and if the margin is not readily made out we dilate the pupil with homatropin.

As soon as the pupil is sufficiently dilated its aphakic portion appears deep black, in contrast with the part that contains the lens, which appears to be of a delicate gray, even when the subluxated lens is perfectly clear and transparent. If light is thrown into the eye by a mirror the normal red reflex is obtained. The portion of the pupil that contains the lens appears thus delicately gray, because light falling on the transparent lens is reflected, especially when in a subluxation it strikes upon its peripheral parts.

The margin of the lens is to be recognized at the sharp line of demarcation between the delicate gray and the deep black of the aphakic pupil. In this case the upper margin is seen; therefore the lens is dislocated downward.

As the margin of the lens comes out still more freely while the pupil is becoming dilated it appears to be brilliantly golden, almost luminous. This is because the rays of light near the margin undergo total reflection. They fall obliquely on the posterior surface of the lens and are reflected back to the observer.

The second method of looking for the margin of the lens is with the ophthalmoscope. It is not always advisable to dilate the pupil in this manner when the lens is luxated, and then we look for the margin with this instrument. Its appearance now is quite different; it looks black. This is because the light returning from the eye is so greatly deviated in its passage by the almost prismatic portion of the periphery of the lens that it does not return to the eye of the examiner, but leaves the margin of the lens unlighted, black; if, however, you will now move your eye slowly toward the opposite side of the margin of the lens, you will come to a place where the margin is resplendent while the rest of the lens is dark, as was shown by *Dimmer*. This is the way in which we determine the direction and the kind of the dislocation. Frequently a distorted and an oblique position coexist. Thus we diagnose in this patient a traumatic dislocation of the lens.

In contradistinction to the traumatic luxation there is a spontaneous luxation which takes place without external cause, concerning which I refer to our studies of congenital dislocation of the lens. We are interested today only in the traumatic dislocation.

When the lens is only shoved somewhat out of the fossa patellaris we call the dislocation subluxation. When the displacement is greater, especially when the lens is no longer completely visible in the pupil, we call it a luxation, but a sharp line cannot be drawn between the two conditions.

How is it that the traumatic dislocation of the lens takes place in an eye which has hitherto been apparently healthy? Normally the suspensory ligament of the lens, the zonule, is tense and fixes the lens perfectly. We can jump and perform gymnastic feats without displacing the lens, yet the zonule is a very delicate structure.

This suspensory ligament can be seen very beautifully if we press carefully on the lens of the fresh eye of an ox, from which the cornea and iris have been dissected away, and it is evident at once that this delicate structure may be torn in a contusion as a result of the increased pressure and displacement of the aqueous.

The subjective symptoms of traumatic subluxation of the lens depend primarily on the extent of the laceration of the zonule. Small lacerations may cause no disturbing symptoms whatever. In rather larger lacerations the lens is movable. It has been thought that an iridocyclitis might be excited by the pulling of a movable lens, but I do not believe in such a mechanical cause of this disease. The patients complain chiefly of disturbances of vision, the essential points of which may be learned from this case.

We ascertained, while the pupil was still contracted and the margin of the lens hardly visible in it, that the patient has a considerable degree of myopia which he says positively that he did not have before the accident. When the zonule is relaxed the lens tries to assume a spherical form, provided that the man is of a suitable age. This relaxation is furnished in traumatic subluxation by the tearing of the zonule; hence the lens has become arched forward and the far point has been brought near. The mechanism of accommodation must be impaired when the zonule is extensively lacerated.

Together with the myopia we find a considerable degree of astigmatism. The explanation of this is that the displaced, or obliquely placed lens refracts light with unequal strength in the different meridians. The refractive power may vary even in different sections of the same meridian.

When the pupil of our patient has been dilated, or when from the first the margin of the lens lies in the pupil and divides it into one portion that does, and one that does not, contain the lens, we have monocular diplopia. Part of the rays enter the eye through the aphakic portion of the pupil, where there is a high degree of hypermetropia and the refractive power of the eye is slight. These rays would meet behind the retina. The rays that pass through the lens undergo a strong refraction, because the lens is extremely convex after laceration of the zonule, and in this part of the pupil the refraction is myopic. In addition to this these rays are deviated downward by the prismatic action of the lens. Thus two images of one point are formed on the retina—monocular diplopia. Not only can the patient see double, but for analogous optic reasons the fundus appears to be double during an ophthalmoscopic examination.

Finally, the subluxated lens may gradually become opaque and so impair vision. This may happen either with or without laceration of its capsule.

Aside from these disturbances, a patient with a subluxated lens must always be in danger that a partial luxation may become total, as the result of the constant movement of the lens and the tearing of the fibres of the zonule. The lens then disappears into the vitreous, where it excites further trouble, or it may be dislocated into the anterior chamber. We must therefore be careful about dilating the pupil in considerable degrees of subluxation.

More often subluxation of the lens causes secondary increase of tension in the eye. This may appear immediately, or soon after the injury; in other more fortunate cases it may not appear at all. We ascribe as the cause of this increased tension a shallowness of the anterior chamber and a closure of its sinus, which in many eyes may suffice to prevent the escape of the aqueous. We see such an increase of tension follow the simple displacement of the lens. If the lens swells as the result of a laceration of its capsule, still more if a spontaneous cataract develops, another factor is added which may lead to increased tension after subluxation.

The duty of the physician in cases of traumatic subluxation of the lens varies with circumstances. The laceration of the zonule cannot be repaired. If the subluxation causes no trouble through a secondary increase of tension and the patient has another good eye it is best to leave it alone. When the injury is fresh the eye should be bandaged for a while, especially when hemorrhage is present. If the patient is much distressed by diplopia a ground glass may be placed before the injured eye to exclude it from participation in the act of vision.

If the other eye has been injured or lost we must try to correct, by means of glasses, the abnormality of refraction produced by the luxation. If one part of the pupil is aphakic this portion may be corrected by means of a cataract glass, or the refraction of the rays that pass through the lens may be corrected by a concave glass; the one of the two that will give the best vision is to be chosen.

If the patient has from the first, or develops in the course of time, troubles referable to secondary increase of tension, the aid of the specialist is necessary. Here, in the clinic, we first try to reduce the tension with eserin. If this cannot be done we perform iridectomy, but experience has taught us that the effect of this operation is not very satisfactory in this form of secondary glaucoma.

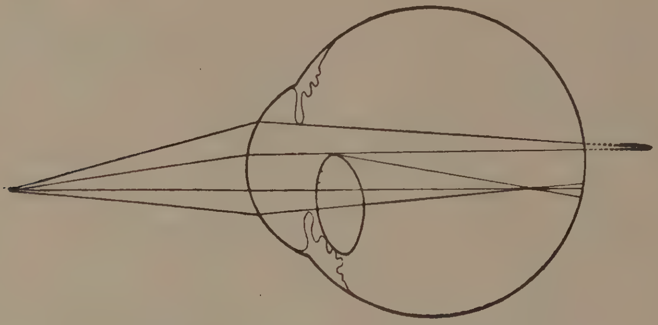

FIG. 73.—Monocular Diplopia in Luxation of the Lens.

Extraction of the lens is difficult, the relaxation and laceration of the zonule readily lead to a great loss of vitreous, and we never attempt to perform this operation except in cases of absolute necessity.

The objective symptoms in traumatic luxation of the lens into the vitreous are in line with those that have been mentioned. The anterior chamber is deep, the iris somewhat sunken, the pupil deep black, the iris tremulous. The same functional hypermetropia exists as after an operation for cataract, if the eye was emmetropic before the injury.

The question what is to be done for eyes in which the lens has been dislocated into the vitreous has been considered in connection with reclination of senile cataract, so here it may be dealt with briefly.

Frequently this form of luxation of the lens is harmless to the eye. The lens usually becomes opaque after a time, swells, shrinks and calcifies; in very rare cases it may be completely resorbed. Even when it has been freely movable for years it generally becomes attached to the retina and chorioid and either grows adherent, or induces secondary glaucoma. Such eyes have to be enucleated finally. An iridectomy alone may be tried at the commencement of the increase of tension, as an extraction of a lens floating in the vitreous is an impossibility.

The lens may also be dislocated into the anterior chamber by a contusion. In such cases the troubles of the patient and the symptoms are quite different from the beginning. The eye has a very marked ciliary injection and the patient complains of severe pains that radiate over half the head. If the cornea is still clear the lens may be seen to lie like a large drop of oil in the anterior chamber, its margin forming a golden, lustrous line. The anterior chamber is deep in its centre, but usually still deeper in its lower portion, where the iris is pressed backward by the lens. The cornea becomes opaque very soon in such cases as these. The gravity of the situation is indicated by the increase of tension occasioned by the direct obstruction to the outflow of the aqueous presented by the lens in the anterior chamber. The lens capsule may remain intact, or it may be torn, in which case, if the cornea is not too opaque, the radiating and irregular opacities of the rapidly swelling lens may be seen very distinctly as it lies in the anterior chamber. The lens does not always remain wholly in the anterior chamber, sometimes it lies partly in it, partly behind the iris, in which case the iris is said to "ride" on the equator of the lens.

The mechanism of displacement of the lens into the anterior chamber as suggested by *Foerster* is as follows:

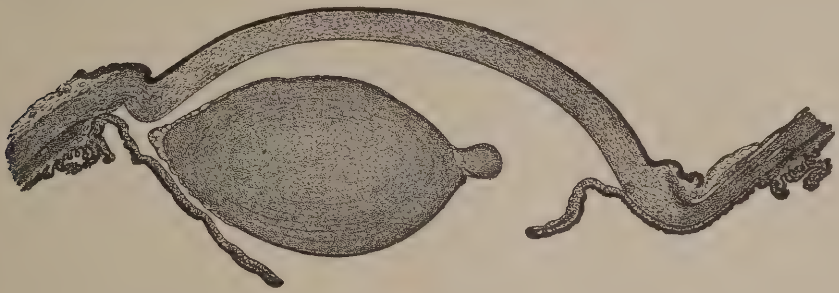

FIG. 74.—Luxation of Lens into the Anterior Chamber.

This form of luxation of the lens is met with only in cases in which the blow falls vertically on the centre of the cornea, so that the cornea is flattened, the aqueous is caused to press the pupillary portion of the iris firmly against the lens and to pouch its peripheral portion back into the posterior chamber, so as to dilate the pupil, perhaps with laceration of its sphincter, and to rupture the zonula. In such a case the lens slips through the dilated pupil into the anterior chamber, the tension of the iris gives way and the pupil contracts, imprisoning the lens in the chamber.

Therapeutically there is only one thing to be done, to extract the lens at once; otherwise the eye may be blinded by secondary glaucoma.

89. Opacities of the Lens after Contusions

This man was struck by a piece of wood in his right eye a year ago. He had a moderate degree of pain and states positively that after the swelling of the lids had subsided he could see well with the eye. About three months later his vision became bad. His claim for indemnity thus far has been rejected because the visual trouble appeared so long after the accident.

One eye is healthy, with perfect vision. In the other, which was formerly injured, an opacity of the lens may be seen both by oblique illumination and with the ophthalmoscope. The pupil should be dilated to facilitate the examination. Then a radiating figure may be perceived with the ophthalmoscope, which is plainly due to the sectorlike arrangement of the lens fibres and appertains to the posterior cortex. This localization is easily determined by means of the parallactic movement. Lenticular opacities following contusions present many clinical pictures, they are met with in the anterior and posterior cortex, as well as throughout the lens. The stage of the cataract, the nature of its etiology and the age of the patient influence the clinical picture.

The question is whether a cataract can develop after a contusion without rupture of the eyeball, or a dislocation of the lens, a long time after the receipt of the injury. It has been proven by experiment and by clinical observation that this question may be answered unconditionally in the affirmative. Many researches have been instituted in which opacities of the lens have been produced in animals after various forms of contusion, but above all clinical observation on man, as shown by many reported cases, has rendered positive the occurrence of these cataracts.

The etiology of such a contusion cataract may be twofold. In the first place the lens may become cataractous after a contusion without the slightest demonstrable injury of its capsule. We must therefore suppose that the concussion of the lens alone is able to bring about a certain displacement in its texture and an injury to its epithelium and cortical fibres, which react with an increased permeability for water.

In the second place it is a noteworthy fact that a laceration may take place somewhere in the capsule of the lens, as the result of a contusion, without luxation of that organ, or rupture of the globe. The posterior capsule, which is particularly thin near the posterior pole, is ruptured more readily than the anterior by a contusion, and many such cases have been reported, but ruptures of the anterior capsule also occur.

It is supposed that the rupture of the capsule takes place in the following way. When the eyeball is compressed from before backward in a contusion its transverse diameter must be increased, the corneoscleral margin with the insertion of the zonule must be stretched, and then the zonule, or the lens capsule may be torn. If the ophthalmologist sees the patient soon after the injury, if the ruptures in the capsule are large, and especially if they lie near the anterior or posterior pole of the lens, he is able to find them without difficulty; but if they are very small, are covered by the iris, or if they lie near the equator of the lens, it is sometimes very difficult, if not impossible to determine clinically whether the contusion cataract occurred with or without a laceration of the capsule. We thus come to the conclusion that the contusion was the cause of this patient's cataract, and will aid him in the maintenance of his claim.

It is nothing uncommon that our patient first observed his visual disturbance several weeks after his injury. How many people discover late in life that they can see better with one eye than with the other! The commencement of the cataract may date back farther than is stated by our patient, for he naturally did not notice the disturbance of vision until the opacities extending from the periphery of the lens had begun to encroach upon the pupil.

In this form of cataract we wait quietly at first, because we know that the opacities may clear up extensively, especially if they are situated in the posterior cortex, but if they extend, as they seem to be doing in this case, extraction must be considered later. *Vossius* was the first to describe the *circular opacity of the anterior surface of the lens,* which corresponds to the form of the pupil, as occurring after contusions. The iris is occasionally imprinted by the compression upon the anterior surface of the lens, and leaves behind a stamped impression of the margin of the pupil. These opacities have a brownish tone of color and consist partly of pigment that adheres to the capsule of the lens.

I will now demonstrate the way in which a contusion cataract is produced in a rabbit. The eye is cocainized and I strike it with the finger, as in percussion, for several minutes, as recommended by *Hess*. The pupil becomes contracted, the iris becomes very hyperæmic and finally an exudate is formed in and covers the pupil. The contusion is repeated to accelerate the development of cataract and I will exhibit the animal at another time.

The first opacities in the lens can usually be seen after a few hours at the posterior suture. Circular opacities also develop in the posterior cortex near the equator of the lens. The experiment is without serious effects to the animal, as this contusion cataract undergoes involution in a few days or weeks.

90. Aseptic Perforating Wounds of the Eye and What the General Practitioner Should Do in Cases of Injury

What is the general practitioner to do when a case of recent injury to the eye enters his office?

The history is to be taken first. A few hours ago a knife blade struck this patient in the eye; he immediately felt severe pain and also felt a warm fluid escape from his eye, since which he has been unable to see well with it.

The next question is whether the injury is superficial, a contusion, or a perforating wound, the latter of which is always the most important.

The lids are to be separated carefully. In the cornea may now be seen a clean cut wound running outward from its centre and extending some millimetres beyond the corneoscleral margin. The margins of the wound in the cornea are swollen and slightly gray. This is because the corneal tissue eagerly absorbs water as soon as the epithelium or endothelium is injured. Where the wound lies in the sclera it is surrounded by a subconjunctival hemorrhage. This is a case of perforating wound of the eyeball.

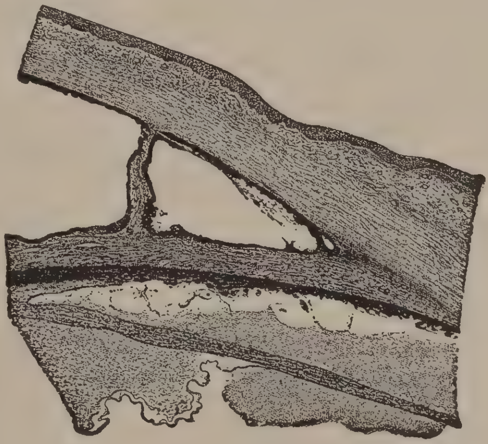

FIG. 75.—Anterior Synechia.

In addition to the history and the appearance of the margins of the wound the signs of perforation are:

1. The condition of the anterior chamber; in fresh cases the anterior chamber is abolished, or very shallow, because the aqueous has escaped.

2. The condition of the intraocular tension; if we let go the margin of the lid and take the tension we find it very low and the eyeball feels soft.

3. The condition of the lens; an injury of the capsule furnishes another positive sign of perforation.

4. The distortion and prolapse of the iris; this furnishes in our case another positive sign. A black brown mass is to be seen between the margins of the corneoscleral wound. Part of the iris was carried out by the escaping aqueous and became caught between the margins of the wound, forming a prolapse of the

iris. When the wound gapes still more widely in these cases a portion of the ciliary body and of the chorioid may prolapse. Even the retina is sometimes visible as a gray cord between the margins; or a clear bead of vitreous may appear and begin to grow turbid in a day or two. In a wound through the sclera traces of blood, or even the penetrating foreign body, may be perceived with the ophthalmoscope. Then when it has been ascertained whether fingers can be counted or not the examination is finished.

The fate of the injured eye depends on whether the wound is, or will become, infected, for however severe a wound of the eye may be it will heal just as well as in other organs, and the preservation of form and vision depends only on how much the interior of the eye has been harmed by hemorrhage, loss of vitreous and injury to the lens, and in how far the cicatrization of the aseptic wound may lead to atrophy. An aseptic wound of the cornea unites very quickly, the swelling and opacity of its margins pass away in a few days; the eye becomes pale in from eight to ten days, and the wound leaves only a scar in the cornea.

The question whether the wound in this case is already infected, whether the foreign body has inoculated some agent of infection in the margins of the wound, or in the interior of the eye, cannot yet be determined. Primary infection of the wound cannot be prevented and will show itself in a few days.

Besides primary infection secondary infection of the wound needs to be borne in mind. Even the normal conjunctiva contains bacteria that may enter the wound subsequently. This danger is particularly great when the conjunctiva is in a state of chronic inflammation, as is frequently the case in people of the laboring classes, or when a dacryocystitis is present. Patients may also touch their injured eyes with their fingers, or with soiled cloths. The danger of secondary infection is great and many eyes injured in this manner are lost in consequence. When this secondary infection has taken place we cannot regard the wound. Evidently prophylaxis cannot be undertaken too early to prevent this complication and as such cases, especially in the country, go to the general practitioner it is plainly his duty to guard against infection.

When any other organ is wounded and bleeds it is to be bandaged. It is remarkable how many practitioners are timid about dealing with an eye, and how many patients with such an injury come to the clinic without even the necessary protective dressing. The general practitioner should not only apply a dressing to such an eye, he can and should treat it operatively.

First the vicinity of the eye, which is often dirty, should be cleansed with soap and water, then with benzin. Then the patient should be laid on the operating table, or on the sofa in the office, and his eye cleansed together with the wound. Some drops of cocain are to be instilled and the conjunctival sac irrigated thoroughly with a sterile physiological salt solution, or with a solution of boric acid. The region of the wound is also to be cleansed.

If the wound is small and none of the contents of the eye are between its margins some sublimate vaseline should be introduced into the conjunctival sac, an antiseptic moist dressing applied and the patient sent either to the clinic, or to bed. If the wound is large and gapes it is best to introduce a suture in the conjunctiva at the corneoscleral margin in order to prevent a further prolapse of the vitreous.

If, as in our case, the iris, chorioid, or vitreous is prolapsed through the wound the eye must be thoroughly cocainized. A small pair of forceps and of scissors should be boiled and some sutures prepared. The patient is then to be prepared, the physician cleanses himself and is then ready to abscise the prolapse. The lid elevator is introduced, the prolapsed iris seized with forceps, drawn out very carefully and cut away with the scissors. If the physician has a steady hand and some iris still remains between the inner lips of the wound he may try to replace it in the anterior chamber, provided that the wound in the cornea has not yet closed. One or more sutures should then be introduced through the conjunctiva bulbi at the corneoscleral margin, including the upper layers of the episclera. The eye is then dressed and now for the first time is the patient ready for the journey to the clinic. Many eyes that have been injured owe their preservation to the stitch taken in time. This therapeutic procedure does not exceed the competence of the general practitioner, and every specialist will be thankful for such cooperation in the treatment of wounded eyes.

In the clinic, still other measures are taken in these cases. Frequently it is necessary to introduce fine sutures in the cornea itself, or to cover the wound with a flap of conjunctiva in order to guard against infection, or to promote a better union.

If the wound in the cornea lies in the limbus the conjunctiva may be slid over it, if it extends farther into the cornea it may be covered by a bridgelike flap of the conjunctiva bulbi with a double pedicle. In many cases we have to make a timely removal of the swollen lens material.

91. Perforating Wounds of the Eye with Acute Purulent Infection and Their Treatment

I always try to impress the point that the first question of the practitioner when he sees an injured eye should be instinctively: Is this a perforating wound, or not? Then follows the second question, whether the wound is infected or not, for of all the dangers incident to an injury of the eye, infection, with its consequences, is the complication which is of the most importance to the prognosis. We fear nothing so much as the entrance of microorganisms into the wound and the infectious inflammation they excite. Every wound of the eye, whether superficial or penetrating, may become infected.

Most superficial injuries of the eye, that become important through infection, lead to ectogenous infectious diseases of the cornea in the form of ulcers, which have already been described.

Every infected perforating wound of the eye has from the start a more serious importance than a superficial infection, for three reasons: 1, an infected perforation, even when the wound is very minute, may very easily cause a total loss of vision; 2, the gravest changes in the form of the eyeball may be brought about, usually by contractions, after the subsidence of the intraocular inflammation; 3, a certain group of infected perforating wounds may not only portend, but bring about grave danger to the second eye through sympathetic inflammation. These are sufficient reasons why no physician who sees an infected perforating wound should neglect to institute correct measures. There are three fundamental types of the clinical picture, between which are many intermediate forms. 1, acute suppuration of all the contents of the eyeball: panophthalmitis; 2, purulent inflammation confined to the anterior segment of the eyeball: ophthalmia purulenta anterior; 3, purulent inflammation confined to the vitreous: ophthalmia purulenta posterior, or abscess of the vitreous.

Two days ago this man was struck in the eye by a splinter of wood. Today both eyelids are very œdematous and swollen, he cannot open his eye, nor can it be opened except by means of the elevator, with which the lids can be separated somewhat, enough to reveal a very grave purulent infection of all the contents of the eyeball.

We find a wound in the cornea, the margins of which are dirty yellow, the aqueous is quite turbid, the lens is opaque and swollen, the iris exhibits a dirty discoloration, the conjunctiva is very chemotic, the eyeball is protuberant, in a condition of exophthalmos, and is immovable. The patient complains of extreme pain and his temperature is 38°.

The quick development of this entire grave clinical picture in one or two days after the injury is characteristic. In such a case there is not the least doubt that it is an acute purulent infection of the interior of the eye following perforation. Pus agents of the most virulent kind must have been transplanted into its interior. In some cases their entrance is more gradual. Thus, for example, a panophthalmitis may originate from a perforating ulcer of the cornea when the suppuration passes inward.

Should such a case of panophthalmitis be left to itself, or should the patient not consent to the removal of the eye, its spontaneous course is as follows: After a number of days, when the inflammation has reached its acme, the pus seeks an exit and escapes, either through the wound, or if this has closed, or is too small, through a rupture at some point in the sclera. The pain ceases as soon as the pus has escaped, but the eye undergoes atrophy, phthisis bulbi.

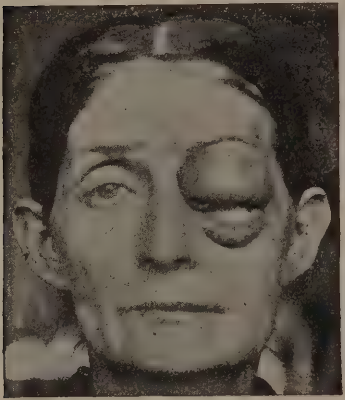

FIG. 76.—Panophthalmitis. Case from the Greifswald Clinic, also in *Axenfeld's* Textbook.

In our second case the eye has likewise a great conjunctival and ciliary injection, but both the great swelling of the lids and the chemosis are wanting. In the cornea we see a flaplike, irregular, perforating wound, the margins of which are not simply slightly gray and swollen, as when it is fresh and aseptic, but infiltrated with yellow pus. The opacity in the parenchyma extends far beyond the limits of the margins of the wound. The anterior chamber is present, the pupil much contracted, the details of the iris are very indistinct and nearly a third of the anterior chamber is filled with a fibrinopurulent exudate. In this case the lens is intact. This is a purulent infection of the wound caused by a perforation of the cornea, which appears to be confined to the anterior segment of the eyeball.

The next clinical picture is still different. This child has been shot with an arrow. The eye is somewhat photophobic and is not greatly irritated, aside from a moderate ciliary injection. At the outer corneoscleral margin is a very small perforation in which the iris is caught. The latter is only moderately discolored and the lens is still transparent. By oblique illumination a yellowish greenish reflex is obtained from deep in the eye, especially when it is looking downward and outward. A weak, reddish light may be seen with the ophthalmoscope when the eye is directed upward. This is a perforating wound of the sclera with prolapse of the iris, but the injury is not aseptic, there is an abscess in the vitreous which as yet is circumscribed.

In order to understand these cases of purulent infection after perforating injuries we must consider how the infection takes place.

An infection may be primary or secondary. The inflammatory agents may be inoculated immediately into the wound by the object that produces the injury. They may be directly adherent to this object, or they may be caught up by it from the surface of the eye, the skin, or the margin of the lid, and carried with it into the eyeball. As I have repeatedly said, under normal conditions and still more when there is inflammation of the conjunctiva, margin of the lid, or lacrymal sac, infectious germs are to be found on the surface of the eye, on the conjunctiva, on the margin and on the skin of the lid.

The other possibility is that the wound was originally aseptic, but that inflammatory agents settled and proliferated in the wound secondarily, after the lapse of hours, days, or even weeks. The larger the wound, the longer it gapes, or is not closed, the greater is the danger of secondary infection. Contusion of the margin of the wound, or prolapse of the iris and vitreous, likewise favors subsequent infection of the wound.

Adherent and ectatic cicatrices may excite a grave internal inflammation of the eye even months or years after an injury has healed aseptically. In cataract operations the resultant scars with incarceration of the iris and cystoid cicatrices may give rise to purulent intraocular inflammation, even to the worst form of panophthalmitis. The same result can come from the scars left by accidental injuries. *Wagenmann* has shown that the purulent inflammation is caused in such cases by a fresh ectogenous infection which begins in the cicatrix. A slight defect in the epithelium over such scars, which may easily be produced mechanically, as by rubbing the eye, may suffice to furnish an entrance to the microorganisms. The rapid penetration of the infectious agents into the vitreous is explained by the peculiar anatomical condition of such eyes and the frequent inclusion of the iris between the margins of the wound.

Such an inflammation may be confined to the anterior segment of the eyeball, or from the first to the posterior segment, the vitreous, and many transition forms between these two possibilities are met with, for the inflammation may be acute or may involve the different parts of the eye during a prolonged period.

We will now turn to the microscopic study of cases in which the inflammation has involved the anterior segment of the eye.

The margins of the wound are swollen and much infiltrated with pus cells. We see very characteristic changes in the anterior chamber. We see a fibrinopurulent inflammation of the iris in which the ciliary body is greatly involved. The tissue of the iris and ciliary body is much thickened and permeated with leucocytes. The vessels are distended with blood, which has escaped from them here and there. A fibrinous exudate, permeated with pus cells, lies on the surface of the iris, in the pupil and on the posterior surface of the cornea.

Sometimes a distinct hypopyon can be seen at the bottom of the anterior chamber. When the purulent inflammation is more severe, or when it has extended farther into the eye, a purulent, fibrinous exudate is thrown off by the ciliary body, which fills the posterior chamber, envelops the lens and even involves the most anterior portion of the vitreous.

It is plain that such an infection as this may lead to the loss of the eye, but we have both clinical and anatomical proof that a purulent or fibrinopurulent inflammation of the anterior segment of the eyeball may recover. If, for example, a purulent infection of the cornea comes to a standstill, the purulent or fibrinopurulent inflammation of the anterior segment of the eyeball may disappear completely. In this connection it may be recalled that after an ulcus serpens has run its course it frequently happens that some posterior synechiæ alone remains.

Recovery may take place after moderately severe cases of suppuration in the anterior chamber, but organic exudates in which vessels have developed usually remain on the anterior and posterior surfaces of the iris, in the anterior and posterior chambers and in the pupil. The exudates in the posterior chamber produce more or less extensive adhesions between the iris and the anterior surface of the lens. The iris itself is generally very atrophic. The inflammation is then past, but even this degree of recovery may be accompanied by sequelæ, of which I will mention only seclusion and occlusion of the pupil with the complications they produce, which has been studied under diseases of the iris.

The results of the graver forms of purulent inflammation of the anterior segment of the eyeball are far more serious. Under the microscope we find extensive layers of connective tissue about the ciliary body that produce adhesions between it, the iris, and the lens, and may encapsule the latter. The nutrition of the lens is thus interfered with and a cataracta complicata develops. If the lens was wounded at the time of injury the suppuration penetrates its substance, its tissue breaks down, while a marked infiltration of pus cells and a folding of the capsule are to be seen. The contraction of the vitreous causes the so called vitreous detachment; the retina becomes detached, frequently torn from the ora serrata, and the final result is phthisis bulbi. True bone tissue and calcification may develop in such organic exudates.

When the purulent inflammation attacked the vitreous first, or when it extended to the vitreous from the anterior segment of the eyeball, it can be perceived that the toxic products of the pus agent caused a massive immigration of pus cells into it, together with a large excretion of fibrin. These pus cells came principally from the ciliary body and retina, both of which are markedly infiltrated and have deposits of exudates on their surfaces. In some cases we meet at an early stage with a swelling and infiltration of the head of the optic nerve. The infiltration of the vitreous may be compared directly with the hypopyon. The phenomenon is most striking and noteworthy that in such cases of purulent infiltration of the vitreous due to infection with the ordinary germs, the chorioid is scarcely involved in the inflammation. Evidently the toxin cannot reach the chorioid well because the retina intervenes. This is to be seen most distinctly in the microscopic preparations of eyes in which the retina was detached at an early period. When the retina has been destroyed in places, or when it remains lying close to the chorioid, the latter becomes involved in the inflammation. Infiltration of its innermost layers and exudations in the suprachorioidal space follow, but the purulent exudate in the vitreous comes chiefly from the retina.

In graver cases of total acute suppuration the vitreous is filled with bacteria and the changes are not confined to the interior of the eye. The toxines induce, by distant action, inflammatory exudations in the retrobulbar tissue and in the vicinity of the organ. This is the anatomical reason of the œdema of the lids, the exophthalmos, and the immobility of the eyeball, met with in panophthalmitis. It goes without saying that such an eye must be blinded. The outer wall of the eyeball may be broken through in panophthalmitis, a quantity of the purulent contents may be evacuated and the eyeball be destroyed by atrophy; phthisis bulbi is the final outcome after panophthalmitis. Milder degrees of purulent infection of the vitreous are met with. On this slide we see that the pus is confined to a portion of the vitreous, forming an abscess. It is surrounded by a marked fibrinous infiltration of the vitreous, by which the infection is localized.

Vision is saved in only the rarest instances of abscess of the vitreous. The abscess is encapsuled by organized connective tissue, the pus is resorbed and replaced by dense connective tissue. Ordinarily the result is worse, even though the inflammation gradually declines. The organized tissue surrounding the abscess is frequently connected with the capsule of the eyeball and as it shrinks it draws the retina away from the subjacent tissue. Usually a considerable detachment of the ciliary body and of the chorioid is also met with. At first, while the processes of healing are taking place, the form of the eyeball is still normal, but very soon the expert notes from the lowering of the intraocular tension that the gradual contraction of the eyeball has begun. With the reduction of the tissue of the vitreous and the onset of total detachment of the retina the fate of such an eye is decided. Perception of light is always uncertain, the projection is bad; phthisis bulbi is the most frequent result of an abscess of the vitreous. A deep depression of the scar after grave injuries is often a distinct sign of a commencing phthisis of the eyeball.

A physician has to decide in such cases as these what part of the responsibility he is able to assume himself and what he must turn over to the specialist. If he is content to turn the injury over to the ophthalmologist I still advise that he thoroughly cleanse the vicinity of the eye with soap and water, and benzin, that he irrigate the conjunctival sac with boric acid solution to remove as much as possible of the purulent secretion, and that he abscise, after cocainization, any tags of tissue that may be hanging from the infected wound, or from the eyeball in which the infection is supposed to be present. Then, after the introduction of an ointment containing sublimate and cocain, or of an antiseptic powder like iodoform, a dressing should be applied and the patient may go his way to the specialist.

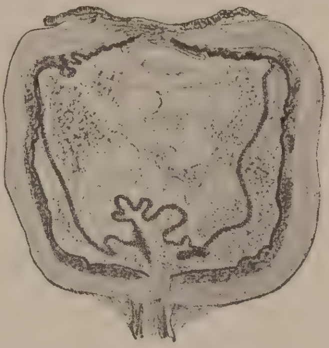

FIG. 77.—Bulbus Phthisicus Quadratus, after a Perforating Ulcus Serpens. Ulcer Cicatrized, Coaptation of the Iris, Empty Lens Capsule. The Lens Has Escaped; Large Abscess of the Vitreous with Detachment of the Retina. Exudate beneath the Retina.

The measures we take vary according to the facts in each particular case, especially according to whether the infection is in the anterior, or the posterior segment of the eyeball. Generally we proceed as follows: In an infected, perforating wound of the cornea associated with iritis and hypopyon we try to ascertain, by slides and cultures taken from its margin, the nature of the infectious agent.

If we find pneumococci, which are not rare, we give the patient one or more injections of pneumococcus serum to immunize the body. If the infectious germ is one against which we have no specific remedy, this measure cannot be taken. Then the wound in the cornea is opened, washed out and the necrotic margins are abscised or touched with the thermocautery in order to destroy the greatest part of the focus of inflammation, if it is within reach.

If there is a severe purulent iritis with a large hypopyon we perform a paracentesis of the anterior chamber at the limbus, if it is not possible to evacuate the hypopyon through the wound. The effect of paracentesis has been described in connection with ulcus serpens.

If the inflammation is not checked by these measures the cauterization and paracentesis must be repeated and the anterior chamber washed out with a sterile 0.85% salt solution. A dressing, rest in bed and free instillation of atropin complete the treatment. I have wholly abandoned the introduction of iodoform into the anterior chamber in purulent infection of the wound, as have other ophthalmologists.

Much benefit may be obtained in suitable cases from covering the defect produced by the abscission of portions of the cornea by a conjunctival plastic operation as done by *Kuhnt*. When paracentesis is no longer indicated, or cannot be performed, we may use subconjunctival injections of salt in order to promote the metabolism in the anterior segment of the eyeball. Whatever happens we do not relax our efforts to save the eye. The iris, zonula and lens, which separate the two portions of the eyeball like a diaphragm, render the passage of the infectious germs into the depths of the eye difficult, and we can do much therapeutically in the anterior segment. Fluid lost from the eye here is readily restored, therefore the prognosis of purulent wound infection in the anterior segment of the eyeball is generally not so bad from the start, and clinical experience has proved that a not unimportant percentage of such eyes may be saved.

Such a perforation is serious when the lens is wounded. The fragments of a swelling lens form excellent nutritive material for suppuration in the eye. In such cases we seek to remove these suppurating, or swelling masses of lens matter in order to disburden the eye.

In cases of primary purulent inflammation of the vitreous we are therapeutically powerless. The measures to be taken depend on the clinical severity of the symptoms. Immediate exenteration should be performed in panophthalmitis.

We assume a serious responsibility when we decide that a purulent infection is situated mainly in the vitreous. In this child the infection of the posterior segment came from a little wound at the limbus. The eye is much injected and has an indistinctness of the iris, but it is not painful and has maintained thus far its normal form. Through the pupil we obtain a yellowish reflex from a circumscribed area. It is always hard to sacrifice such an eye, but on the other hand we cannot make a free surgical incision and evacuate the pus. We must therefore look on rather inactively; we may bandage the eye, we may try inunctions in the hope that in this way the infection may be brought to a standstill and the pus resorbed. We know that in some cases a spontaneous recovery takes place, when the extent of the infiltration and the virulence of the agents are slight, but on the other hand we must not forget that the ultimate outcome of such infections after recovery is phthisis bulbi. Above all we must consider a danger that threatens in such cases. We can never know whether such an intraocular infection may not some day excite a sympathetic iritis in the other eye in spite of its appearance, which is perhaps mild. Most weighty reasons these why we should not try to save eyes with abscess of the vitreous. Therefore, if the injection of the eye, the irritation and the iritis remain unchanged, and the examination reveals loss of perception of light, or a bad projection, we shall ask the parents to consent to the removal of the eye. Even adults frequently need several days to decide upon such a sacrifice.

92. Perforation Cataract with Intraocular Infection

This patient was injured yesterday and presents a very grave condition today. The eye has a marked conjunctival and ciliary injection with great chemosis, and presents in the cornea a wound with swollen and dirty margins. The first question, whether the wound is perforating or not, must unfortunately be answered in the affirmative, for the anterior chamber is almost abolished and has a purulent exudate at its bottom. Our second question, whether the wound is already infected, receives the same answer. The iris looks muddy and its details are indistinct. All this might be explainable by the changes in the margins of the wound, but the opacity of the lens proves that the wound is perforating.

A closer examination of this opacity of the lens reveals that it presents not simply the gray tone of a traumatic cataract, but a dirty yellowish tone of color which leaves no doubt that pus is already present, that this is an abscess of the lens. We have learned from other cases with traumatic cataract that the case becomes more serious with each opening of the lens capsule; when an infection is present in the swelling masses of the lens the prognosis is very grave. In this patient the abscess developed in the lens a few hours after the injury.

This was because the broken down lenticular substance furnishes an excellent nutritive material for the pus agents within the eye.

Anatomical examination of such eyes shows that the purulent inflammation extends very quickly from the wound in the capsule into the lens. The pus agents may be traced on the slide in lanes between the necrotic fibres.

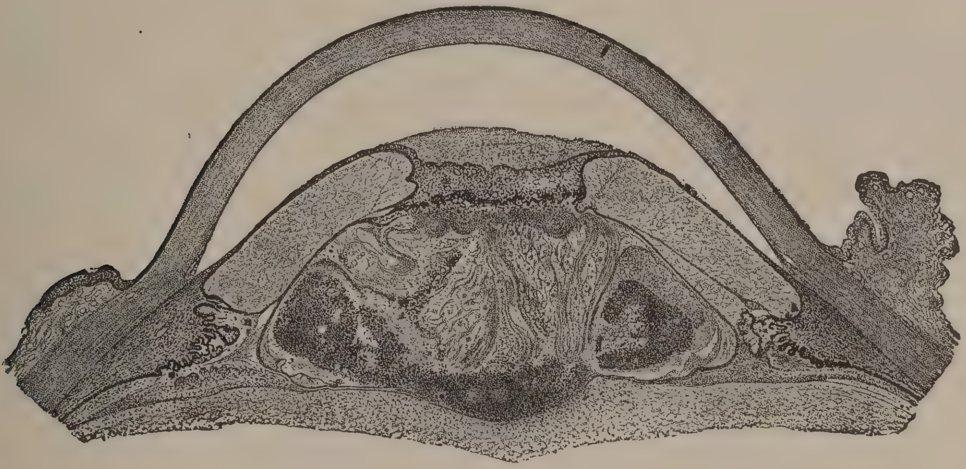

FIG. 78.—Perforation Cataract with Intraocular Infection.

The next question is whether the foreign body is still in the eye or not. When it is positively known to be present we make an attempt to remove it; if it is a splinter of iron the magnet is applied at once. Even when there is no intraocular foreign body we try to save the eye. The general surgeon opens and drains an abscess; we can only try to withdraw the suppurating masses of lens material from the eye and wash out the anterior chamber.

The prognosis depends primarily on whether the posterior capsule has been perforated and the suppuration has already spread into the vitreous. It depends also on the nature of the infection and the power of resistance of the eye, which varies in different individuals. Sometimes it happens that an eye with such an abscess of the lens is saved, but usually the result is panophthalmitis and phthisis bulbi.

Aside from suppuration of the contents of the lens from acute wound infection, we meet with an involvement of the injured lens in the chronic iridocyclitis traumatica induced by perforating wounds. Here is such a case, in which perforation took place several weeks ago. According to the report of his physician a traumatic cataract was produced and removed by operation. The eye is much injected and atrophy is commencing. The patient shrinks from the gentlest touch. Above is a coloboma of the iris produced by operation, the margins of the pupil are adherent to the remains of the shrunken lens, while among the remnants of the lens are numerous dark lumps of pigment and a fibrous tissue permeated with blood vessels, which passes beneath the wound made at the operation.

In the anatomical examination of such eyes it is a striking fact that the tissue in the vicinity, whether the wound be one produced by accident or by operation, tends to proliferate into the sac formed by the capsule of the lens. The eye in this patient demands enucleation.

93. Sympathetic Iridocyclitis and Its Treatment

About eight weeks ago this patient received a perforating wound of his left eye, for which he was treated elsewhere, but after some weeks he discontinued treatment as he needed to go back to work. The injured eye remained painful and an inflammation gradually developed in his right eye, which impaired his vision and finally brought him to us.

The left eye is totally blind and atrophic. A deeply depressed scar in the sclera marks the site of the perforation. The patient shrinks when the eyeball is touched, showing that the ciliary body is tender. The pupil is totally occluded by exudate, the iris is greatly thickened and its details are indistinct.

The right eye presents a ciliary injection, deposits on *Descemet's* membrane and indistinctness of the details of the iris. The pupil has a strong tendency to contract and in one place a posterior synechia may be seen. With the ophthalmoscope the vitreous appears cloudy from minute opacities. Our patient has been so unfortunate as to develop that which is most feared in perforating wounds: sympathetic iridocyclitis of the other eye.

1. What Must Be the Clinical Nature of the Disease in the First Eye when Sympathetic Inflammation Will Occur in the Other?

The fact is somewhat remarkable that after an injury of one eye a disease may appear in the other that exactly resembles the disease in the first. It becomes still more so when we investigate the general condition of such patients and find no other morbid changes in the organism. Our patient is perfectly healthy. He has no fever, or other sign of a general disease. His eyes alone are affected. In such a condition of affairs it is imperative that we should know the clinical nature of the disease in the first eye when sympathetic inflammation will occur in the other. In what diseases of the eye have we to fear sympathetic disease of its fellow? It is beyond doubt of the greatest importance that we should be able to perceive and recognize the sympathetic nature of iridocyclitis.

The first essential is that the capsule of the eyeball first diseased must have been opened. The largest wounds of the eye heal and remain without irritation if they are aseptic. It is also true that sympathetic inflammation need not be taken into account or feared in many cases of intraocular infection after wounds of the eye. When pus agents enter the vitreous of an eye the first tempestuous symptoms of inflammation usually appear within twenty-four hours, at any rate they do not delay for days. The ciliary injection, the pain, the discoloration of the iris, the accumulation of pus in the anterior chamber, or the vitreous, quickly reveal the purulent infection. Either panophthalmitis develops in a short time with great chemosis and protrusion of the globe, or the purulent inflammation runs a more subacute course and ends in phthisis bulbi.

In this case the course was quite different. The healing of the wound was apparently normal, but the eye never became perfectly quiet. Very gradually the signs of a chronic plastic iridocyclitis developed. The eye exhibited ciliary injection, deposits were formed on the posterior surface of the cornea, the iris became discolored, showed a muddy discoloration, according to the report of his physician, the pupil contracted and no longer reacted to atropin, while one posterior synechia appeared after another. About this time large opacities were demonstrable in the vitreous; signs of fibrinous uveitis and cyclitis. Finally the entire pupil became occluded by a fibrinous exudate. This exclusion and occlusion of the pupil are still present. Meantime the eyeball gradually atrophied. This is the clinical picture of fibrinous uveitis, or iridocyclitis. Hence there are two kinds of wound infection of the eye which may follow an injury: purulent uveitis and chronic plastic uveitis.

The second essential for the origin of a sympathetic ophthalmia is that the first eye must have suffered from a chronic plastic uveitis in addition to the injury. This fibrinous iridocyclitis alone is able to excite a sympathetic inflammation in the other eye.

There are exceptions to this rule. In rare cases a purulent inflammation of the uveal tract may be followed by a sympathetic inflammation of the second eye, but we must remember that these two forms of intraocular infection do not contrast sharply, either clinically or anatomically, and that the purulent may pass into a fibrinous inflammation. It is, however, beyond doubt that the notorious pus agents are not the agents of sympathetic iridocyclitis; therefore, if a sympathetic iridocyclitis follows an intraocular wound infection which originally was of a purulent nature, we must suppose that a mixed infection was present.

Thus we have learned how the disease in the first eye must appear in order to have a sympathetic ophthalmia follow in the second eye, and the next question is: Under what conditions are these requirements for the pathogenesis of this disease fulfilled?

The commonest cause of sympathetic ophthalmia is a perforating injury followed by a persistent chronic fibrinous inflammation, which usually leads to atrophy, to phthisis bulbi. Most of the sympathetic diseases of the eye originate from such phthisical, injured eyes.

In rare cases this disease follows a perforating ulcer of the cornea. Such ulcers heal quite smoothly, as a rule, and when a sympathetic iridocyclitis does follow, it must be that the morbific agent entered the eye through the perforation and excited a fibrinous uveitis, which does not otherwise appertain to the clinical picture of a corneal ulcer.

More frequently sympathetic ophthalmia is met with after operations, especially after unfortunate operations for cataract. In these cases also the wound was not aseptic, but a plastic, fibrinous iridocyclitis was produced. In these cases too the opening of the capsule of the globe and the fibrinous uveitis were the preliminary requisites for the origin of sympathetic ophthalmia.

These three factors exhaust the causes of sympathetic ophthalmia. The subconjunctival ruptures of the eyeball alone seem at first to hold an exceptional position. They are generally benign, because the wound in the sclera is covered by the conjunctiva, and when a sympathetic iridocyclitis appears in such a case it would seem at first glance as if an opening in the capsule of the globe was not an unconditional necessity for the genesis of a sympathetic ophthalmia, but it is soon shown that a subconjunctival rupture can excite sympathetic inflammation only when it is associated with inflammation of the uveal tract, and the anatomical researches of *Deutschmann*, *Meyer* and others have proved the source of the intraocular inflammation. It is evident in such cases that small lacerations existed in the conjunctiva, sufficient to permit the passage of the microorganisms, but so minute as to escape clinical observation.

The danger of sympathetic iridocyclitis must be well known, but must not be overestimated. The conditions are by no means such that any injured eyeball in which a fibrinous uveitis develops must produce sympathetic ophthalmia. On the contrary I have demonstrated atrophied eyes in which this danger was not present. In regard to the frequency of sympathetic inflammation our knowledge is not accurate, because atrophic eyeballs, which perhaps harbored the germs of sympathetic iridocyclitis, are frequently and properly removed at the right time. My estimate is that only a small percentage of infected, perforating wounds, from 1 to 3%, result in sympathetic inflammation. More men than women suffer because the former are much more exposed to injuries of the eye. Children, among whom serious perforating wounds are very frequent, furnish a large contingent of the cases of sympathetic ophthalmia. I once collated at the clinic in Halle 264 cases of fresh, perforating injuries of the eye; 64, 25%, of these were in children from 1 to 10 years old.

The clinical picture presented by the iridocyclitis of the injured eye, which gives rise to a sympathetic inflammation, is very similar to that of an ordinary iritis, as may be seen by comparison with this case of syphilitic plastic iritis. We find in both deposits on *Descemet's* membrane, cloudiness of the aqueous, discoloration of the iris with indistinctness of its details, and the question arises how to differentiate clinically the one from the other. Granted that we know what the conditions of the disease in the first eye must be in order to excite a sympathetic inflammation in the other, and that we recognize those conditions as present, do we then know positively that the disease in our patient is sympathetic iridocyclitis? Is it not conceivable that an iridocyclitis from some other cause may start in the second eye independently of that in the first and quite accidentally some time after the injury? In our patient with a phthisical eyeball cannot a plastic, syphilitic, tuberculous, rheumatic, or idiopathic iritis occur just the same as in an eye of a person hitherto well? Who tells us that in this case the disease of the second eye is in such a condition of dependence on the injury of the first that we must call it sympathetic?

It must be admitted that the diagnosis of a sympathetic iridocyclitis is only one of probability. This is because the uvea, the most vascular membrane of the eye, must react in the same way to the entrance of all agents of inflammation. The fibrinous exudates are always the same whether an infectious bacillus, or a micrococcus settles in the iris. Only a few specific agents of infection excite specific clinical pictures in the iris, and these do not in every case. In syphilitic iridocyclitis we sometimes meet with characteristic papules, sometimes with an ordinary diffuse, fibrinous iritis. Sympathetic iridocyclitis is characterized by no clinically specific features and we have as yet no biological reactions by means of which to recognize whether the organism is affected by a sympathetic inflammation. I do not doubt that the unknown agent of sympathetic inflammation is a specific germ, but the specific nature of sympathetic iritis cannot yet be recognized clinically. We need finer biological methods than we have yet at our disposal to detect a certain one from a group of morphologically similar germs, even among those which are well known to us. Hence the diagnosis of sympathetic inflammation can never be made with absolute certainty, a fact that renders its exact study very difficult. Confirmation of the diagnosis should be sought from a thorough general examination of the entire body and an intercurrent iridocyclitis of the second eye from other causes should be excluded by a clinical and serological examination. This necessitates the clinical precept that the blood of a patient having sympathetic ophthalmia should be tested for *Wassermann's* reaction, as has been done in this case.

2. The Lapse of Time Between the Diseases of the Two Eyes

The shortest interval that has been observed between the receipt of the injury and the onset of the sympathetic iridocyclitis is fourteen days; the average interval is from six to ten weeks; beyond this there are no fixed limits. Cases have been reported in which sympathetic iridocyclitis appeared after the lapse of years, and even of decades. The longer the time that has elapsed since the injury the more doubtful is the diagnosis and the more carefully must other forms of iritis be excluded. One thing is certain. Whenever a sympathetic ophthalmia appears after so long an interval the phthisical bulb is in a condition of fresh irritation, is injected, sensitive to pressure and contains fresh foci of inflammation in its uvea. *Schirmer* has called special attention to this. We do not know the origin of this fresh inflammation in the formerly injured eye; it is uncertain whether the old inflammation has rekindled, or whether the agents have laid thus long in quiescence. The theory that the fresh inflammation is excited by the formation of bone, which is often met with in these stumps, was disproved long ago. Ossification never excites irritation in a phthisical eye and its process has nothing to do with an awakening of slumbering germs.

3. The Clinical Picture Presented in the Sympathizing Eye

The clinical details of sympathetic iridocyclitis can be studied much better in the second eye than in the first, which is often phthisical. We may divide cases clinically into mild, moderately severe and grave; if we wish to base the classification on anatomical principles we may divide them into the serous and the fibrinous, but these can never be differentiated sharply and may change from one into the other, as a benign into a malignant form. The attack in this patient should be classed as moderately severe.

This is an iridocyclitis; therefore the first symptoms appear in the iris and ciliary body. In almost all cases the disease begins without any prodromal symptoms, a fact that is of importance. A failure of vision is not rarely the first intimation the patients have of their condition. The first objective sign is a slight ciliary injection, but very soon the disease reveals whether it is mild or grave. The richer the exudations are in fibrin the severer is the attack. In mild cases we find a slight cloudiness of the aqueous, and little gray, or brown deposits on *Descemet's* membrane. The details of the iris are scarcely obscured, the pupil is dilated by atropin, the anterior chamber is deep and the intraocular tension is somewhat increased. Ophthalmoscopically the vitreous very soon becomes cloudy, because of a multitude of minute opacities, and the papilla becomes indistinct, partly because of the cloudy media, partly because of an inflammatory involvement of the nerve itself. This form usually runs a very chronic, but benign course, though it may change into the malignant form.

If the attack is grave and the uveal exudations are rich in fibrin, posterior synechiæ appear very soon in addition to the deposits on *Descemet's* membrane. They may be broken up by atropin, but new ones constantly appear, the pupil resists the atropin more and more and becomes contracted. The fibrin in the anterior chamber accumulates and is deposited in the form of a transient hypopyon. The entire margin of the pupil finally becomes adherent to the lens capsule and then the aqueous can no longer pass from the posterior to the anterior chamber, but bulges forward the middle portion of the iris, giving rise to a crater pupil and to secondary glaucoma.

Still more malignant are the cases in which the exudation is so great that it fills the entire posterior chamber and forms extensive adhesions between the surfaces of the iris and lens. These masses of exudate contract in the course of the next few weeks and create radiating folds in the iris. The iris itself appears to be enormously thickened, the entire pupil is covered by masses of fibrin and both occlusio and seclusio pupillæ are present. Then for a while the picture is that of secondary glaucoma. It is now impossible to see into the eye and perceive the great opacities in the vitreous, even though it could be done at first. Weeks and months elapse before the disease reaches its acme. The deepest layers of the cornea may become opaque as a result of the increase of tension, but the lens is the principal sufferer if the disease does not finally take a favorable turn; its nutrition is cut off by the enveloping exudate and a complicated cataract develops. The fluctuations between improvement and aggravation are characteristic of the grave cases. Finally the intraocular exudate shrinks, hypotony follows the increased tension, ablatio and phthisis bulbi form the final scene. Very malignant cases, which fortunately are quite rare, may begin with chemosis and severe cyclitic pains, but ordinarily the intraocular changes and the pain are not in proportion to each other and severe pain is excited only by the increase of tension. All of these changes, especially the opacities in the vitreous, indicate clinically that the entire uvea is involved, and sometimes, as *Haab* has pointed out, the involvement of the chorioid can be seen with the ophthalmoscope. Small, roundish, reddish yellow spots appear in the periphery of the fundus, extend into the vicinity of the papilla and may be differentiated from other chorioiditic patches by the fact that they have no marked pigmentation. More rarely small, brownish spots are to be seen in the macula. The important point to be learned from all this is that the sympathetic disease is situated in the vascular tunic of the eye. It is a uveitis in which all segments of the chorioid are involved, but which in most cases presents the picture of an iritis, or of an iridocyclitis. This fact is of fundamental importance for the full comprehension of the disease.

It has been maintained, in only a small percentage of the cases, that the sympathetic disease may appear as a pure papilloretinitis. In such cases only a slight papillitis with obscuration of the margins of the disc, opacities and, sometimes, little hemorrhages in the surrounding retina, have been found. As such a papilloretinitis retrograded promptly after enucleation *Schirmer* concluded that the offending microorganisms were present only in the eye first diseased and that their metabolic products had excited the inflammation of the papilla in the second eye by way of the optic nerve.

It was shown by *Becker* that a chorioiditis was demonstrable microscopically in an eye in which only a slight swelling of the papilla had been found with the ophthalmoscope. Even though this so called papilloretinitis sympathica may be independent of a chorioiditis it seems to me that the hemorrhages observed indicate another, not a toxic, mode of origin. I am of the opinion that the papilloretinitis originates hæmatogenously, the same as sympathetic iridocyclitis, but instead of inundating the uvea the agents settle only in the vessels of the papilla, a place in which they cannot be so much at home as in the uvea, and from which they do not excite such grave syndromes. I consider a differentiation of papilloretinitis sympathica as a special form of sympathetic infection to be a priori conceivable, although its independence of the uveitis has not yet been satisfactorily proven. The retrogression of the symptoms after enucleation, as claimed, does not suffice. *Schirmer* believes that this papilloretinitis was produced by diffusion of the toxin from one eye to the other by way of the optic nerve, and disappeared after enucleation of the first eye in a way similar to that in which the iritis associated with ulcus serpens disappears after section of the ulcer, but we know that an optic neuritis often can take spontaneously an almost critical turn for the better. If the sympathetic papilloretinitis was produced by toxins through diffusion this must be a genuine symptom. This localized diffusion along the optic nerve must be quite frequent, and a monolateral neuritis due to some other cause must be able to produce in this way a neuritis in the other eye. More than this, such a diffusion must take place in every sympathetic iritis. But a few pages later, after *Schirmer* has advanced this hypothesis of sympathetic papilloretinitis, he says, in his article on sympathetic iritis, "A transition of toxins from the injured eye, which *Rosenmeyer* thought possible, is excluded from the first," so I cannot discover an adequate foundation for the differentiation of papilloretinitis sympathica. At any rate the sympathetic disease in the second eye of this patient is an iridocyclitis, exactly like that in the first, or wounded eye.

4. Pathology of Sympathetic Iridocyclitis

The principal seat of sympathetic ophthalmia is in the uvea, where the following details may be noted with the microscope:

Throughout the iris, ciliary body and chorioid disseminated groups of mononucleated cells are to be seen, together with a diffuse infiltration of the entire tissue, and a marked fibrinous exudate on the surface of the iris and ciliary body. The iris is greatly infiltrated at the acme of the inflammation, and has masses of exudation on its posterior surface, in the pupil and on its anterior surface. The ciliary body is also uniformly infiltrated with small cells. Great masses of fibrinous exudate proceed directly from it to envelop the lens and extend into the vitreous.

A later stage is shown in another preparation. As these masses become organized, vessels appear and connective tissue cords draw the epithelial coat and the ciliary processes strongly inward. Even the entire ciliary body may be pulled inward and it is self evident that this contraction of connective tissue may finally cause detachment of the retina. The condition in the chorioid is still more characteristic of the inflammation that excites sympathetic ophthalmia. Here we find a great number of inflammatory foci which are situated chiefly in the layer of the large vessels and rather avoid the inner layers. In another preparation it may be seen that the nodes are joined by a rather diffuse infiltration of mononucleated cells, not of pus corpuscles. I wish to call particular attention to the places within the diffuse infiltration in which we find nests that contain epithelioid cells and giant cells. There is a certain resemblance to tuberculosis, but that disease is not present. There is no caseation, and tubercle bacilli have never been found in them. Among others the plasma cells may be noticed. Their offshoots, with finely granular contents, may disappear into the tissue and then, as *Axenfeld* has pointed out, if the granules float out into the tissue they may readily be mistaken for cocci. This error has happened many times. When the inflammation undergoes involution the diffuse and circumscribed infiltrations are followed by an atrophy of the chorioid.

In comparison with this inflammation of the chorioid the lesions in the retina and optic nerve are extraordinarily unimportant. In the retina they are almost wholly secondary, an atrophy of the nervous elements and a proliferation of the supporting tissue evidently occasioned by nutritive disturbance in the chorioid. The lesions in the papilla and optic nerve are of more interest because of the relation they bear to the pathogenesis of the entire disease.

We find an œdematous swelling of the papilla which is particularly marked when the inflammatory infiltration of the chorioid extends into its neighborhood. Single foci of inflammation are absent, except that the vessels are sometimes surrounded by mantles of round cells. In the trunk of the optic nerve the region of the lamina cribrosa may be more densely infiltrated with round cells and inflammatory lesions may also be found in the inner sheath. The endothelial cells of the arachnoid trabeculæ may be proliferated. In other cases no infiltration of round cells is to be found in the optic nerve. One fact is now certain: The degree of inflammation decreases the farther peripherally we go in the optic nerve toward the chiasm and even in the immediate vicinity of the papilla the inflammatory lesions do not correspond in the remotest degree to those in the uvea. All this is of importance to our conception of the genesis of sympathetic iridocyclitis.

5. Treatment and Prognosis of Sympathetic Iridocyclitis

The first questions that arise after sympathetic iridocyclitis has once appeared are: What influence does the first eye exert over the course of the infection in the second, and what disposition shall be made of the injured one? When the injured eye is totally blind and atrophic the decision to enucleate immediately is easily made. It is much more difficult to determine what influence is exerted on the course of the inflammation by the enucleation of the exciting eye. From the nature of the case it is conceivable that the inflammatory agents in the second eye may not be influenced thereby, whether other germs are found in the first or not. Neither a sudden check, nor a turn for the better takes place even when the enucleation has been performed immediately after the outbreak of the inflammation, but it must not be forgotten that the primary focus of infection, from which came the invasion and from which fresh reinforcements of inflammatory agents may constantly be sent forth, is destroyed by the enucleation. In this way may be explained the beneficial influence ascribed to enucleation of the primarily diseased eye by many ophthalmologists.

When the primarily injured eye retains even a trace of vision the indications for treatment are quite different, for under no conditions should such an eye be removed. It may be saved with a useful degree of vision after the second eye has been totally lost, and even when its functions are insufficient the question of its enucleation should be considered very maturely.

Treatment of the Sympathizing Eye

It is evident from the difficulties that beset the entire subject that a patient with sympathetic iridocyclitis should be treated in a hospital where the measures calculated to preserve the second eye can be carried out, after enucleation of the first.

The fundamental principles of the local treatment of the sympathizing eye are the same as those of the treatment of other forms of iritis. Atropin is instilled regularly and an attempt is made to break up the posterior synechiæ. In the graver cases this means is insufficient to prevent exclusion of the pupil and the onset of increased tension. When this increase of tension is due to a serous iritis eserin may be tried for a while, but, according to my observation, the value of meiotics in such cases is rather illusory. Heat is applied to the eye for hours during the day by means of the thermaphore, in order to increase the effect of the reactive hyperæmia. It may be said in favor of subconjunctival injections of salt solution that, by their stimulating effect, they aid in the resorption of masses of exudate from the anterior chamber, that they contribute to the clearing up of the vitreous, and that in many cases they have reduced an increased tension to normal. On the contrary the subconjunctival injection of mercurial preparations has been abandoned.

In former times many attempts were made to obtain improvement by operative intervention, but the results must have been wretched and the general consensus of opinion today is that no operation should be performed on an eye in the active stage of a sympathetic iridocyclitis. Even when there is a secondary increase of tension an early iridectomy is contraindicated, because the apertures created are occluded at once by exudates produced by a rekindling of the inflammation, or because in the presence of broad adhesions the iridectomy is technically without result. At the most sclerotomy may occasionally be considered when the danger from secondary glaucoma is very threatening. Every critical clinician will admit that in the graver cases we must frequently be resigned to watch the course in the diseased eye. The question whether an operation may not be necessary for optical reasons does not arise in any case until the inflammation has subsided completely and the eye has been quiet for months. Such an operation may be either an iridectomy or an extraction of the lens. The condition most favorable for an iridectomy is an exclusion of the pupil without total posterior synechiæ. In such a case the iris is not seized at the pupillary margin, because we wish to avoid laceration of the adherent lens capsule. Cases have been operated on successfully by the extraction of the transparent lens. When extraction of the lens is necessary in such an eye *Wenzel's* method is the best to employ. A curved incision is made in the limbus of the cornea, passing the knife through the iris and the lens. After the lens matter has been removed a triangular piece is excised from the thickened tissues composed of the iris and lenticular capsule by means of two converging incisions from the angles of the wound.

General Treatment

In addition to the local treatment an energetic general treatment is indicated, and for many years inunctions of mercury and sweating have been employed for this purpose. The strength of the patient must be taken into account in both of these methods.[1]

Even if it is true that cases of sympathetic ophthalmia were much more serious than they are now, the prognosis of this disease remains sufficiently grave. Sympathetic iridocyclitis is a fate to awaken dread and sympathy. Physicians have an important duty to perform in its prophylaxis by the exercise of correct judgment concerning injuries of the eye.

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[1] [Good results from the use of very large doses of sodium salicylate have been reported.—*F.*]

94. Genesis of Sympathetic Ophthalmia and Its Prophylaxis

Sympathetic ophthalmia is a disease that is prevented much more easily than cured. In this respect the duty of the general practitioner is one of great responsibility.

I. Genesis of Sympathetic Iridocyclitis

Unfortunately there is little known positively concerning the origin of this disease, its pathogenesis is almost completely enveloped in darkness. According to clinical experience it is only when the specific agents of infection have penetrated through a wound in the capsule of the globe into the interior of the eye and have produced there a chronic uveitis, that the second eye may suffer from the same disease. The appearance of the agent of sympathetic ophthalmia, the predisposing conditions that influence the disease, and the paths by which the pathogenic agents enter the organism and reach the second eye, are still wholly unknown. We could therefore deal briefly with the pathogenesis of sympathetic ophthalmia were it not that our therapeutic and prophylactic measures are influenced by the theories advanced concerning its origin.

(*a.*) *The Pathogenic Agent of Sympathetic Iridocyclitis*

Every possible germ has been accused of being the agent of sympathetic iridocyclitis. *Deutschmann* in particular believed the staphylococcus to be the agent. We need not go into the details; all the assertions lack proof and the agent still remains completely unknown. There are only two hypotheses that can be drawn from clinical experience concerning its nature; it must be one of the morbific agents that retain their vitality for a long time in the eye and in the organism, for otherwise the long duration of the sympathetic inflammation, its tendency to recurrence, and the fact that it may appear many weeks after the commencement of the disease in the first eye, cannot be explained. It must also be a germ that is not pathogenic to the same degree to other organs of the body as to the eye; indeed it is highly probable that it is not infectious to any other part of the body. Thus far we have obtained no evidence that sympathetic ophthalmia can occur in the lower animals, so we have to suppose that man alone is susceptible to this pathogenic agent. Much clinical experience has taught us that persons who are suffering from sympathetic ophthalmia exhibit no lesions in other organs even approximately resembling those in the eye, or any signs of a general disease. It should be noted particularly that such patients show no symptoms that can be ascribed to a meningitis.

There is no doubt that there are infectious agents which are specially adapted to and have a specific affinity for the eye. Trachoma is an example. The agent of trachoma, with which we are as yet unacquainted, excites the gravest pathological changes in the eye without infecting any other organ and without disturbing the general condition. I have proved that it cannot be demonstrated serologically whether or not the organism is under the influence of a trachomatous infection. The diplobacillus discovered by *Morax* and *Axenfeld* also excites a specific infectious disease, which may lead to grave corneal ulcerations, on the human eye alone, but cannot infect other animals. No one who will not acknowledge this specific affinity of the agent of sympathetic iridocyclitis for the tissue of the human eye can give a plausible explanation of the pathogenesis of this disease. Without this hypothesis the phenomenon that the second eye alone becomes diseased while all the other organs of the body remain unaffected cannot be explained. With it must deal all theories concerning the occurrence of sympathetic iridocyclitis.

(b.) Predisposition to Sympathetic Iridocyclitis

None of the conditions that appertain to predisposition, none of the complicated processes of susceptibility which favor the development of disease, are yet known to us in the slightest degree, even in those infectious diseases in which we are acquainted with the agent. Those that favor predisposition to sympathetic iridocyclitis are equally unknown. We know only that a perforating wound is necessary and that a plastic uveitis must always be present. The wound may be large or small, the eyeball may be atrophic, or may have retained its form. Uveitis may develop after many infections without the production of a sympathetic iridocyclitis. How often the specific agent enters the eye and what conditions determine whether a disease in the second eye shall, or shall not be excited, we do not know. It may occur at any age, though it is naturally met with most frequently in those periods of life when perforating wounds of the eye are the most common. Children furnish a large proportion of the cases of perforated wounds and also of sympathetic ophthalmia and from what we have learned otherwise from the study of infection it seems likely that they are predisposed to sympathetic iridocyclitis by other factors than their liability to injury.

The importance ascribed by ophthalmology to the influence of nervous transmissions has been altogether too great, and sufficient consideration has not been paid to the other numerous factors which must be taken into account in infectious diseases to explain congenital and acquired predisposition. For a long time the efforts of the ophthalmologist were devoted to attempts to demonstrate the etiological connection of the nervous symptoms of irritation to the inflammation. All of these attempts to ascribe a governing influence to the ciliary irritation in the pathogenesis of sympathetic ophthalmia are nothing else than gropings in the field of predisposition, for the optic nerve at first, the ciliary nerves later, according to *Heinrich Mueller*, were supposed to conduct irritative conditions in a peculiar way into the other eye in order to call forth in it also symptoms of irritation which were supposed to gradually increase into inflammation, but there is no such thing as an inflammation called forth by nerve irritation alone, which has such grave exudations as are present in the first eye. Both the agent and the nature of the predisposition to this disease are unknown to us.

(c.) The Way in Which Agents Are Disseminated in Sympathetic Iridocyclitis

Three theories exist in regard to the way in which the morbific agents of sympathetic iridocyclitis pass into the second eye: 1. The migration theory of *Leber* and *Deutschmann;* 2, the modified cilioneural theory of *Schmidt-Rimpler;* 3, the metastasis theory of *Berlin* and *Roemer.*

1. Leber-Deutschmann's Migration Theory

Leber, who was the first to affirm the infectious nature of the sympathetic inflammation in both eyes, assumed that the disease reached the second eye by way of the optic nerve. According to this theory the agents are not transported passively, but the inflammation actively creeps forward in the continuity of the tissue and in the lymph channels of the optic nerve, in other words they excite and advance by a lymphangitis. This theory was maintained by *Deutschmann,* who believed the agents in both eyes to be staphylococci, a view that has been proved to be erroneous. Although the advocates of this theory assert that it does not interfere with the migration theory, yet the latter can make no claim to furnish the fundamental explanation of sympathetic ophthalmia, because in the first place it contravenes important clinical and anatomical symptoms in the clinical syndrome, and, in the second place, it is contrary to the doctrine of infection. We will first consider it clinically. It is admitted by all that the assumption that the eye first diseased suffers from an infectious uveitis is not the only factor to be considered in the migration theory. Even the rare occurrence of sympathetic iritis in panophthalmitis cannot be explained well by the migration hypothesis. In such cases all clinicians must admit the presence of mixed infections in the eyes first affected. Nothing could lead the migration theory to require a certain time for the migration from one eye to the other, except the purpose of explaining the fact that the earliest appearance of the inflammation in the second eye is fourteen days after the injury; but the interval between the diseases of the two eyes is explained altogether differently from the standpoint of bacteriology.

The cases in which sympathetic ophthalmia has appeared after opticociliary resection are also inconvenient for the migration theory. The path of the microorganisms to the second eye is closed by the resection, and auxiliary hypotheses are again needed. The cicatrix produced by the resection is impermeable for dead substances in dead animals, but permeable in living animals for leucocytes containing particles of coloring matter.

The difficulties of the migration theory increase when we wish to explain the clinical fact that the second eye may become affected three or four weeks after the enucleation of the first. The advocates of this theory have said that the agents had entered the optic tract before the enucleation was performed. In that case the pathogenic agents that maintained a grave, plastic inflammation in the eye must change their character essentially and suddenly in the lymph sheaths of the optic nerve.

A clinical fact absolutely unexplainable by the migration theory is that we never meet with clinical symptoms of a meningitis in the course of a sympathetic ophthalmia, although, according to this hypothesis, a lymphangitis must take place at the base of the brain, especially about the chiasm. There is nothing in this theory to prevent the microorganisms at the chiasm from wandering from the intervaginal space to the meninges and exciting there an inflammation, and, if such an inflammation should be only half as severe as that in the first eye affected, the clinical symptoms of such a meningitis could not escape us, but clinical observation shows that this region must be regarded as healthy during the course of a sympathetic iridocyclitis. If the agents of sympathetic inflammation extend along the optic tract by continuity, as assumed by this theory, without noticeable inflammation of these parts, their character as inflammatory agents must have undergone considerable modification. Such a vital change within the lymph channels does not take place, at least in staphylococci, for it is contrary to what is known of the pathogenicity of these pus agents that they should excite a severe inflammation in an eye, suddenly lose their power as excitants of inflammation in the lymph channels of the optic nerve and the base of the brain, and then regain unabated vigor in the other eye. It must not be forgotten that staphylococci are more infectious to man than to any animal used for experiment. If the slight rubbing of these agents into the skin suffices to produce a severe furunculous inflammation, what must be expected at the base of the human brain if virulent staphylococci wander by continuity from the eye to the meninges!

It is therefore of interest to us to know how this portion of the brain appears in men who suffer from sympathetic ophthalmia. Several such cases, in which the patients died of intercurrent diseases, have been examined. The important fact common to all was that as the distance from the eye increased the inflammatory symptoms in the optic nerve decreased, and were absent at the chiasm. I have no doubt that the inflammatory changes in the first part of the optic nerve in sympathetic ophthalmia are distant effects of the intraocular inflammation. At any rate, we can in no way conclude, from the condition of inflammatory infiltration in the retrobulbar portion of the second nerve, that inflammatory agents have passed along its lymph channels toward the eyeball. The advocates of the migration theory have maintained that the fundamental principle of the parasitic inflammation advancing in the continuity of the optic nerve in sympathetic ophthalmia does not rest on the question whether the staphylococci mentioned by *Deutschmann* are the agents or not, but this objection does not hold good, because the basis of the migration theory rests upon experiments on animals with staphylococci. We must deny from the very beginning that any conclusions can be drawn as to the pathogenesis of human sympathetic ophthalmia from experiments on animals with any pus agents, for sympathetic iridocyclitis is not a purulent inflammation. I have therefore taken this position: "The migration theory cannot be authorized to draw from experiments on animals conclusions as to the symptoms of sympathetic ophthalmia in man, or to explain the pathogenesis of the same through a migration of germs along the optic nerve, until a continuous growth of such pathogenic germs as are dangerous to the eye, but do not endanger the rest of the organism through a general infection, has been demonstrated by it along this track from one eye to the other." This postulate has not been complied with by the migration hypothesis, for when we review the experiments in which morbid agents were found after intraocular infection it appears that masses of germs were taken into the blood, but were not deposited by way of the optic nerve at the base of the brain. I have made numerous experiments with this result. Now the migration theory may assume that it has to deal with altogether different specific agents. As I have already said, such a postulate is indispensable, but when the advocates of this hypothesis adopt the idea that the pathogenic agents are specially adapted to the interior of the eye they must drop their hypothesis. The eyes only are involved in sympathetic ophthalmia, because evidently the agents of that disease develop and increase in them alone. If a segment lies between them, especially a portion of the base of the brain, which is healthy, it follows that it can contain no inflammatory agents. Then it has been claimed that the agents of sympathetic iridocyclitis may remain in an indifferent condition so far as the lymph channels and the base of the brain are concerned, but such an auxiliary hypothesis is untenable. Under such a presumption a migration of the agents of sympathetic iridocyclitis by continuity is completely illusory, for in that case inflammatory agents may develop in living tissue without exciting inflammation. This is not true, and the idea finally adopted by the advocates of this theory, that the agents of sympathetic iridocyclitis do not find good nutritive media in the meninges directly contradicts the hypothesis of a continuous lymphangitis extending from one eye to the other.

Finally the migration theory does not explain why the sympathetic ophthalmia begins in the iris and ciliary body in the overwhelming majority of cases. If it were a lymphangitis that extended from one eye to the other every sympathetic ophthalmia should begin as a papillitis, but it does begin as a uveitis. Here again the hypothesis must seek a way of escape. Some have conjectured that trivial changes in the optic nerve could have been present in such cases. While elsewhere the migration theory always speaks of a process that extends by continuity, here the agents arrive at *Tenon's* space from the sheath of the optic nerve without causing inflammation, skip an intermediate space and reenter the eyeball with the ciliary vessels.

Not only is the migration theory unacceptable for all these clinical reasons, but I have shown by experiment that no intraocular infection corresponds to the premises of the *Leber-Deutschmann* hypothesis. Intraocular infections, whatever they may be and however produced, never excite a lymphangitis confined to the optic nerve, which creeps along from one eye to the other by way of the chiasm. If virulent bacteria are employed they enter the circulation en masse and the animal dies of general infection. If less virulent stocks, or bacteria of a saprophytic nature, are used they do not extend to the optic tract, certainly not along it from one eye to the other. *Leber-Deutschmann's* migration theory is untenable and can no longer be employed as the basis of the explanation of the pathogenesis of sympathetic ophthalmia.

2. The Modified Cilioneural Theory of Schmidt-Rimpler

The second theory is the one advocated by *Schmidt-Rimpler*, which assumes that the irritation of the ciliary nerves in the eye first affected induces a susceptibility to the disease in the other, through a reflex disturbance of its circulation and nutrition. The ciliary irritation in the first eye simply prepares the soil in the uvea of the second for the settlement of whatever bacteria may come from a bodily disease. No distinction is made whether the question is one of bacteria, of toxines, or of both. *Panas* added to this obscure conception the idea that general toxic influences, such as alcoholism, menstrual troubles and catarrhal diseases of the throat and nose, were predisposing factors to sympathetic iridocyclitis. This theory is likewise untenable, because it is at variance with the clinical facts and is readily disproved by experiment.

In the first place, according to this theory, sympathetic ophthalmia can attack only persons who are already sick, in whose blood bacteria circulate, or have entered from some pathological condition, but if anything is clinically certain, it is the fact that perfectly healthy persons suffer from sympathetic ophthalmia.

In the second place, this hypothesis does not explain why a certain form of chronic plastic uveitis, the peculiar nature of which has been demonstrated by *Fuchs*, must always appear in the eye first diseased. Why cannot glaucoma and other conditions associated with marked ciliary irritation likewise cause sympathetic inflammation, if it depends wholly on the excitation of ciliary irritation?

The hypothesis is also unable to explain the characteristic interval of time between the infection of the first eye and that of the second. Why must at least fourteen days elapse after the injury before the onset of the sympathetic inflammation?

Again, how can this hypothesis explain the rare occurrence of sympathetic ophthalmia after panophthalmitis, in which extreme ciliary irritation is certainly not lacking?

Above all it is absolutely impossible to understand from *Schmidt-Rimpler's* hypothesis how sympathetic ophthalmia can appear several days, or even weeks, after enucleation of the first eye, when there has been no ciliary irritation for a long time.

How can the modified cilioneural theory explain the clinical cases in which sympathetic inflammation appears with no symptoms of irritation? We certainly see sympathetic inflammation begin without irritation, especially in serous uveitis, so that the first trouble noticed by the patient is the reduction of vision. The explanation of the pathogenesis of sympathetic iridocyclitis cannot be based on a hypothesis which does not account for such a large number of characteristic signs.

In addition to this the experimental foundation of this theory is a failure. Certain investigators, such as *Mooren* and *Rumpf*, and *Jessner*, decided from insufficient experiments that when the exposed iris of one eye was irritated the amount of albumin and fibrin in the aqueous of the other eye was increased. We know that the composition of the aqueous changes in inflammations in the anterior segment of the eyeball because the dilated vessels of the ciliary body become permeable to the constituents of the serum, and if the claim made had proved correct the modified cilioneural hypothesis would have received at least some support, but *Wessely* has shown with great exactitude that the amount of albumin in the aqueous of the second eye remains perfectly normal when the first eye is irritated.

I have proved experimentally myself that no intraocular inflammation in the first eye is able to influence by reflex action the amount of antibodies contained in the aqueous and vitreous of the second eye. Even section of the trigeminus is unable to induce by reflex action an increase of the albumin, or a change in the content of antibodies in the aqueous of the second eye. Therefore, the premise that the reflex transmission of irritation can excite disturbances in the circulation and nutrition of the second eye has not been proved and cannot be proved. Recently an attempt has been made to vindicate this hypothesis, but, to my mind, with very little success. *Schmidt-Rimpler* thinks that the site of the irritation is especially in the ciliary body, and that the investigation of the aqueous does not suffice, because the latter is not secreted by the ciliary body, according to modern views. In the first place, this assumption that the hypothetical irritation is confined to the ciliary body and is not transmitted to the iris is an improbability, for the ciliary nerves supply both the ciliary body and the iris. In the second place, I have proved that the composition of the intraocular fluid behind the iris of the second eye, the secretion of which by the ciliary body is not contested by *Schmidt-Rimpler*, is not influenced in the least by an intraocular irritation of the first eye.

The advocates of this hypothesis have performed surprising evolutions in order to try to evade these facts. While they formerly relied on the experiments of *Rumpf* and *Mooren*, they now claim that it is not necessary that any change in the composition of the aqueous should be produced by the hypothetically transmitted irritation. By this claim they have so modified the theory that it may be abandoned, for such irritations cannot be traced and tested. To maintain that irritation of one eye induces a lacrymation of the other does not touch upon the question whether an infectious disease of the second eye is favored, much less caused, in this manner, and this is *the* question in sympathetic iridocyclitis. The latest turn is the statement that animal experiments contradict *Schmidt-Rimpler's* theory, but that in man a transmission of the irritation to the other eye may still be able to act in loco. Animal experiment is insufficient for demonstration, because it is impossible to produce in animals the nervous irritation of the second eye known as sympathetic irritation. Many things are confused in such conclusions. In the first place the symptom complex of sympathetic irritation has hitherto been considered as having nothing to do with sympathetic inflammation. In the second place, it is not necessary that the symptom complex of the so called sympathetic irritation should appear in animals, for, in my opinion, this symptom complex does not form a true ophthalmological picture, but pertains to traumatic hysteria. The question is whether a purely reflex irritation that originates in the first eye can influence the processes of circulation and nutrition in the second so as to favor, or cause, a settlement of microorganisms from the blood. There is no positive evidence in favor of such a conception even in man, and I am opposed to the idea, because it is too untenable and may lead to far reaching consequences, as may be shown by an example.

The question has been raised what part traumatism plays in the etiology of hereditary syphilitic keratitis parenchymatosa. It has been claimed not only that a superficial injury may cause the disease in the eye directly injured, but that this traumatic disease of the first eye may pass by way of the vasomotor irritation to a locus minoris resistentiæ in the second eye, and there cause an outbreak of the keratitis.

The real is united with the unreal in this theory. It is certainly true that the settlement of bacteria circulating in the blood is favored by an injury to the tissue. We know this to be true of osteomyelitis and tuberculosis. But that a slight injury to the nonvascular cornea induces the settlement of spirochætæ in it is as yet unproved, and *E. v. Hippel* has rightly spoken in opposition to such an idea, and has insisted that hereditary syphilitic keratitis may occasionally appear coincidentally with an injury without any etiological connection between them, inasmuch as the disease attacks the two eyes one after the other, without any prodromal symptoms of irritation, in thousands of cases. The idea that the injury of the cornea of the first eye may cause the infection of the second with spirochætæ is purely theoretical. If it is correct, there is no disease of the eye that cannot be ascribed to an accident. Who can tell the limit of such a hypothetical reflex action of irritation? It is only necessary to rub the eye with a handkerchief and any iritis that may appear a few weeks later in the other eye may be ascribed to the injury of the first by the aid of a hypothetical nervous or vasomotor irritation. This is the logical consequence of the modified cilioneural theory and of the fruitless attempts to maintain it. The hypothesis has not advanced our knowledge of the hæmatogenous infectious diseases of the eye, it causes nothing but confusion. The sooner we divest ourselves of this theory, the better it will be for the progress of our science. We need not expect from Nature so unsuitable a contrivance as one which makes the nutrition of one eye dependent on conditions of irritation in the other. I, at least, perceive in the embryological development of the human pair of eyes a strong indication in the fact that, in spite of the close nervous connection, by means of which the two eyes perform their functions together, the nutrition of each is independent of that of the other.

3. Sympathetic Iridocyclitis as a Specific Metastasis

There is only one theory known at present which gives a natural explanation of all the clinical symptoms of sympathetic iridocyclitis and receives adequate experimental support. This theory is that sympathetic iridocyclitis originates hæmatogenously, just the same as any other spontaneous iridocyclitis, and is due to a specific metastasis from the eye first diseased. *Mackenzie* suggested this theory, but *Berlin*, in 1880, was the first to enunciate it clearly. *Berlin* assumed that the agents in question entered the general circulation from the eye first affected, lodged probably in various places in the organism, but found conditions which rendered their renewed development possible only in the other eye. This hypothesis attracted little attention at first, but I have brought it into prominence by extensive experimental researches, and I can prove that it furnishes an adequate explanation of all the symptoms in the pathogenesis of sympathetic iridocyclitis.

I will consider first the objections that have been raised to the transmission of the disease through the formation of metastases. It is difficult to explain by any other hypothesis why a minimum interval is necessary for the origin of a sympathetic iridocyclitis, but the fact that the onset of a sympathetic ophthalmia takes place after a certain time of incubation is plausible from our views concerning metastasis. The plastic uveitis begins after the lapse of days or weeks. It is also premised that the infection takes place from isolated agents, but isolated agents have little prospect of causing a metastasis. It is necessary that these agents shall have increased and have overcome the resistance of the living tissue before they can force their way into the blood. Even in pyæmic conditions weeks often elapse before the agents can break into the blood channels.

Our theory also explains how it is that sympathetic iridocyclitis can break out some weeks after the enucleation of the wounded eye. We know that microorganisms, when they have once entered the circulation, may retain their vitality for a long time in the internal organs, and the idea that the unknown agent of sympathetic iridocyclitis may retain its vitality for a long time in the body needs no discussion at the present time, when the investigation of protozoa is in active progress, when we know that trypanosomata which have apparently disappeared may reappear after many weeks.

The question, why the second eye only is affected while the rest of the body remains well, was formerly urged vehemently against the conception of sympathetic iridocyclitis as metastatic, but forms no objection to the student of bacteriology. No one who is familiar with the specific adaptation of a series of microorganisms, and with the elective affinities of bacterial products to certain constituents of the protoplasm can find anything more wonderful in the localization of the agents of sympathetic iridocyclitis in the second eye, than in the affection of the human blood cells by the plasmodia of malaria in a certain stage of development. In order to understand sympathetic iridocyclitis we need only to remember that there are microorganisms that may settle in all kinds of tissue, and other infectious agents which may dwell in only one form of cell.

Moreover, I know of no theory which is better fitted to explain the clinical fact that sympathetic iridocyclitis may appear without any prodromal symptoms of irritation. Just as the iridocyclitis may develop in the first eye, after the minutest injury, without previous irritation by any other process, so the sympathetic ophthalmia may develop in the second eye without prodromal symptoms as soon as the specific infectious agents have been implanted in it by way of metastasis.

Our theory becomes still more probable from the fact that it is able to furnish a natural explanation of the absence of meningitic symptoms, and of the commencement of the inflammation in the iris, or the ciliary body, in the uvea. When we assume that the infection is carried to the second eye by way of the blood the absence of symptoms referable to the base of the brain is clear at once, particularly if the microorganisms are such as are dangerous only to the eye.

The best opportunity for the agents to settle in the second eye would be afforded in all probability by the organs most abundantly supplied with blood, the uvea, in which the inflammation usually starts. The peculiar nature of the subdivision of the vessels and the smallness of the capillaries may perhaps play a part in this. When the inflammation really begins as a papillitis is it not just as conceivable that the agents may occasionally settle in the capillary system of the optic nerve? At any rate it is shown by the subdivision of metastases in the same organ that quite specific relations exist between the place of settlement and the bacteria. I need only mention the exemption of the kidneys in the tuberculosis of guinea pigs, the specific glanders of the same animal, the lepra nervorum of man, and the localization of the agents of acute articular rheumatism in the joints.

At the present time the pathogenesis of sympathetic ophthalmia is best explained by the theory of specific metastasis, and I may repeat here some points which I propounded on a former occasion: "It has hitherto been thought possible that a syphilitic iridocyclitis, or a syphilitic papillitis, may originate through the entrance of infectious germs from the blood, but something else has been looked for in the origin of sympathetic iridocyclitis, chorioiditis and papillitis. It seems to me to be an advantage to our theory that it places sympathetic iritis, iridocyclitis and papillitis in the same group as other genuine forms of iritis and iridocyclitis that originate in the absence of injury, so far as pathogenesis is concerned. Thus we come to understand why the diagnosis of a sympathetic ophthalmia is often one of probability." The clinical fact that we cannot calculate upon the occurrence of this disease is likewise readily explainable by the idea of metastasis. If there were a lymphangitis along the path of the optic nerve, which always had to travel the same way, if a reflex transmission of irritation had to first prepare the soil for a settlement, rather more uniformity would be expected in regard to the time of the outbreak of the sympathetic disease than is the case; on the contrary its appearance cannot be calculated on, and this is customary in metastasis.

The question now arises whether this theory, even though it may harmonize naturally with the clinical facts, receives experimental support. Such support needs the proof of three facts.

First, it must be proved that after an acute or chronic intraocular infection a part of the pathogenic agents do in fact enter the blood. I have demonstrated this in a great variety of intraocular infections. A portion of the morbific agents enter the circulation from the interior of the eye and are deposited in the large glands not only in infectious inflammations that lead to sepsis, but in all other intraocular inflammations as well.

Second, it must be proved that the agents that enter the blood after intraocular infections invade, each according to its nature, with a certain predilection, those organs in which they excite only their characteristic disease. I have demonstrated this also. For example: when the as yet unknown agents of the aphthæ epizooticæ are injected into the vitreous of suitable animals, they enter the blood, seek out their specific localization and produce a specific foot and mouth disease. Hence we are justified in believing that the agents of sympathetic iridocyclitis may likewise find from the blood suitable quarters in the vascular regions of the eye alone.

Finally it must be proved that pathogenic agents which excite inflammations only within the eye and are indifferent to the other organs of the body, may not only be found in the internal organs after an intraocular infection, but can also be traced to the iris of the other eye. This may be demonstrated by the following experiment: If a dense culture of spores of the bacillus subtilis is injected into the vitreous of several rabbits, through the cornea and lens so as not to injure the blood vessels, and if the animals are killed at different times, culturally demonstrable spores of the bacillus subtilis are to be found in the internal organs of all the rabbits and in the iris of the second eye of many of them. The final condition alone, to produce after an intraocular infection of the first eye a similar inflammation of the second, the animal remaining meanwhile in perfect health, i.e., to produce a sympathetic ophthalmia, cannot be complied with, because we do not yet know the agent of this disease.

II. Prophylaxis of Sympathetic Iridocyclitis

I have given this brief course of instruction as to the present position of science concerning the origin of sympathetic ophthalmia because the major part of our prophylactic measures have been, and always will be, influenced by these theories. Sympathetic iridocyclitis is difficult to cure when it has once become established, but it will not appear if timely precautions are taken to render the focus of infection harmless.

Anyone who believes in the modified cilioneural theory must logically advocate the preventive opticociliary neurotomy, in order to prevent in this way the reflex transmission of irritation and so remove the predisposing cause of sympathetic ophthalmia. Likewise anyone who adheres to the migration theory must try to destroy the path followed by the wandering germs by a preventive opticociliary neurectomy. Both of these operations were in fact highly recommended formerly. They were based on the principle of dividing all of the posterior connections of the eye first diseased in order to isolate it. The optic nerve, ciliary nerves and vessels are simply divided in neurotomy, in neurectomy a piece of the optic nerve is also removed. Clinical experience has proved that neither operation has any prophylactic value, but that sympathetic iridocyclitis may appear after either. Such a result is not surprising to those who believe that the agents may break their way into the blood channels after overcoming the resistance of the tissues. I have proved experimentally that the microorganisms in intraocular infections can be taken into the blood from eyes after opticociliary resection just as well as from normal eyes.

A second prophylactic measure is exenteration of the globe, but this likewise has proved not an absolutely certain method of preventing the disease in the second eye. Modern researches have shown that little traces of the uvea may remain and form the source of a metastatic inflammation after an exenteration. When the case is one of panophthalmitis there is, as a rule, no choice but to perform a careful exenteration.

The only certain prophylactic measure to prevent sympathetic iridocyclitis is the timely enucleation of the injured eye, when the presence of an inflammation that is able to give rise to a sympathetic inflammation is respected. The intraocular focus of infection is removed with certainty by an enucleation, but, as we cannot know in any given case whether or not any of the agents have already been carried away we can consider the prevention sought for by the enucleation as attained only after the second eye has been free from inflammation for at least four weeks after the operation, as *Schirmer* has correctly insisted.

The precept to perform a timely operation on an eye which is suspected to contain a uveitis that may give rise to sympathetic ophthalmia is not always to be easily complied with in practice, especially when the injured eye still retains some vision and the hope remains that the traumatic uveitis will recover. Cases of this nature must be treated by specialists. The duty of the general practitioner is easier; he has only to take care that wounded eyes in which uveitis has developed after the injury are sent in time to the ophthalmologist. It may happen that a patient will refuse to submit to the operation; in such a case it is advisable that he attest in writing that he assumes the responsibility himself. As an assistant I have seen the Professor of Ophthalmology himself not proof against a suit for damages, although he had been thoroughly conscientious and had given timely advice to have the enucleation performed.

95. Foreign Bodies in the Eye and the Extraction of Splinters of Iron with the Magnet

Our patient is a smith, into whose eye something flew a week ago, while he was engaged in his work, beating iron with iron. He felt a severe pain for a short time. At first he could still see, but for the past three or four days the eye has been blind. The eye is unirritated and exhibits a small, linear cicatrix almost in the centre of the cornea. The anterior chamber is normal. The lens is completely opaque, and a small fissure can be detected in the anterior lenticular capsule.

Our first question in every injury of the eye, whether the wound is perforating, or not, is answered by the fact that the lens is opaque, that there is a traumatic cataract. The second question, most important as regards the fate of the eye, whether infection is present, fortunately can be answered in the negative, for the eye is absolutely unirritated and is free from all signs of inflammation.

In many cases of injury we have to answer a third question, whether the foreign body is still present in the eye. It may be asked a priori: Why is this last question of such great importance? What difference does it make if a minute splinter in an aseptic condition has penetrated into the eye and become fixed there? Can it not become encapsulated without causing reaction?

Both experiments and much clinical experience have proved that there is scarcely a single kind of foreign body which can be borne in the eye without harm. With few exceptions eyes that contain foreign bodies lose after a variable length of time both their function and their form.

I. Tolerance of the Eye to Intraocular Foreign Bodies

The foreign bodies most often met with are bits of iron, steel, copper, stone, glass, porcelain, splinters of wood, shot, grains of powder and particles of sand. We know, from *Leber's* researches concerning the origin of inflammation, that even the purely aseptic foreign bodies do not remain unchanged in the eye, but that small parts of almost all are dissolved by the intraocular fluids, spread by diffusion, and excite inflammation through their chemical actions. The degree of the inflammation depends on the chemical constituents of the foreign body, and we can therefore differentiate between those that excite only a slight reaction and those that produce the most serious changes. For example: if a small, aseptic splinter of glass, porcelain, or wood is driven into the eye it will produce at first only a slight degree of inflammation; it may become encapsuled, and the results attendant on the cicatricial contraction, disturbances of vision and detachment of the retina, may not appear until considerably later.

The situation is entirely different when a bit of copper has been driven into the eye, as by the explosion of a percussion cap. Every bit of copper excites a destructive, purulent inflammation in the eye. If the piece of percussion cap is in the anterior chamber a purulent iridocyclitis develops, if it has penetrated into the vitreous it shortly becomes enveloped by a large collection of pus, which necessitates the enucleation of the eye. In the lens alone minute particles of copper may be borne for years and produce no bad effects, aside from the formation of a cataract.

Particles of iron are the foreign bodies most commonly met with in practice. What would become of the eye of this patient if it contains a splinter of iron, as we suspect it does, and if the foreign body should be allowed to remain there? Perhaps what I said on this subject when discussing diseases of the lens may be recalled. An aseptic bit of iron may be borne apparently without reaction for a long time, but gradually the iron becomes dissolved by the carbonic acid in the tissues of the eye, within which the dissolved iron spreads by diffusion; this leads to a series of important clinical and anatomical changes.

The first clinical sign of this intraocular effect of iron is that the eye begins to be hemeralopic; vision is bad in the twilight. The visual field may also be concentrically contracted and finally the central vision fails, because of a progressive retinal atrophy that results from the effect of the iron upon the ganglion cells and nerve fibrils of the retina. When it is possible to see into the eye a peculiar pigmentation of the retina, which resembles that of retinitis pigmentosa, may be seen extending from the periphery. This is followed by peculiar changes in the iris and lens. The iris assumes a brown, or greenish appearance. The lens presents the signs of iron cataract whether the foreign body is in it, or in the vitreous; it receives a brown discoloration, and rust brown points appear along the pupillary margin, consisting of proliferated capsular epithelium containing deposits of the iron compound. Finally, opacities appear in the vitreous. The injury produced on the uvea by the action of the iron is especially shown by recurrent hemorrhages. This symptom complex we call siderosis bulbi. Its anatomical basis has been studied particularly by *E. v. Hippel.* The final result is enucleation.

II. Demonstration of a Foreign Body in the Eye

How are we to determine whether a foreign body is still present within the eye? We must first ascertain if we can see the foreign body. Foreign bodies in the anterior chamber are naturally the most easy to detect, but the angle of the chamber may form a hiding place, or they may have penetrated deeply into the parenchyma of the iris. Eyelashes are not very rarely carried into the anterior chamber at the time of the injury, and these may lead to the development of cysts of the iris.

We have certain points to guide us when the foreign body is in the vitreous. If it passed through the cornea we can see the little wound, or scar. The symptoms of a perforation, abolition of the anterior chamber after the escape of the aqueous, and reduction of the intraocular tension, usually are met with only just after the injury, because when the wound closes quickly the intraocular tension is soon restored; they may be altogether absent when the perforating splinter is very minute. We therefore try to follow the track of the foreign body. When it has passed through the lens we look for wounds in the anterior and the posterior capsule, which may be connected by a straight line, though the splinter may turn and deviate from a direct course.

If we find nothing to guide us in the still transparent lens we turn to the iris. No prolapse takes place when the splinters are small, but the pupil is frequently drawn somewhat toward the place of perforation, or we may see in the iris the aperture through which the foreign body passed. The place may also be indicated by a small hemorrhage.

If the foreign body entered the eye through the sclera we may find little hemorrhages in the vicinity of the wound, or a portion of the vitreous, or of the uvea, may be present between the lips of the wound. If the refractive media are not opaque we make an examination with the ophthalmoscope.

The force of the foreign body is usually so great that it does not remain near the wound of entrance, hanging in the vitreous, in which it sinks more or less, but is driven forward to the opposite wall of the eyeball, where it either remains sticking in the membranes of the eye, or rebounds more or less and then sinks. It may even pass through the opposite wall of the eyeball and escape into the orbit. This possibility must be borne in mind, particularly when we are dealing with shot wounds of the eye.

The ophthalmoscopic findings differ very much. The foreign body can frequently be seen, looking like a black point. Sometimes splinters of iron have a metallic lustre, but usually they are just as dark as bits of stone, or other foreign bodies. A splinter of copper often gives a yellowish red reflex from the depths of the eye, especially when it can be seen by focal illumination. Transparent bits of glass cannot be seen, but sometimes we may observe a cord-like opacity that marks the track of the foreign body, while the latter may be enveloped in dense opacities, or hemorrhages. In other cases the place where the foreign body struck the wall of the eye can be recognized; it has a whitish appearance and is often surrounded by hemorrhages. If the retina and chorioid were lacerated the white sclera is bared, or the margins of the laceration of the retina may be perceived. If the foreign body has been in the vitreous for a long time it may be encapsuled, or enveloped in a fresh inflammatory exudate, so that only whitish, or yellowish masses may be seen; as a rule they are situated in the lower part of the eyeball.

In still other cases we may see bubbles of air in the vitreous; these have clear centres and deep black edges. Their presence proves that the posterior segment of the globe has been opened, but we can infer nothing from them as to the presence of a foreign body. If the refractive media are still clear and the vision is still good, the position of a foreign body may be located by a scotoma in the visual field, which is caused by the injury to the retina. The foreign body may, indeed, occasionally be perceived entoptically by the patient himself. In many cases, however, the ophthalmoscopic diagnosis of a foreign body is rendered difficult by opacities and hemorrhages in the vitreous, and when the lens is totally opaque, as in this patient, the hope of seeing the foreign body is vain from the start.

Our next resource is the X ray examination. We take an X ray picture of the eyeball first in one position and then in another, and may in this way determine the location of the larger foreign bodies within the eye. It is particularly important that this method be used in the examination of shot wounds, in order that we may be sure that the shot is not still within the eye. Small splinters are apt to escape detection by this method, because they do not cast sufficiently sharp images.

Many such eyes must be left to their fate, unless they contain one kind of foreign body where it cannot be seen: a splinter of iron. It has been calculated that 75% of all foreign bodies that penetrate into the vitreous are composed of iron or steel. We have two more methods at our disposal with which to demonstrate the presence of such foreign bodies.

If we suspect that a splinter of iron is within the eye of a patient, we employ, with the necessary precautions, the sideroscope, which was devised by *Asmus* for ophthalmological purposes. This instrument consists of a magnetic needle hung on a thread, which is brought close to the eye, so that the movement of the needle will indicate the presence of the minutest bit of iron. In order to read the indications of the movements of the needle *Asmus* uses *Gauss'* mirror detector. A little mirror is attached to and moves with the needle, so as to reflect a scale placed horizontally in front of it, the images of which are read through a telescope. We can utilize the varying amplitude of the excursions of the needle at different places on the eyeball to locate the position of the foreign body. The presence of splinters of iron that weigh less than one milligram can be demonstrated by means of this delicate instrument.

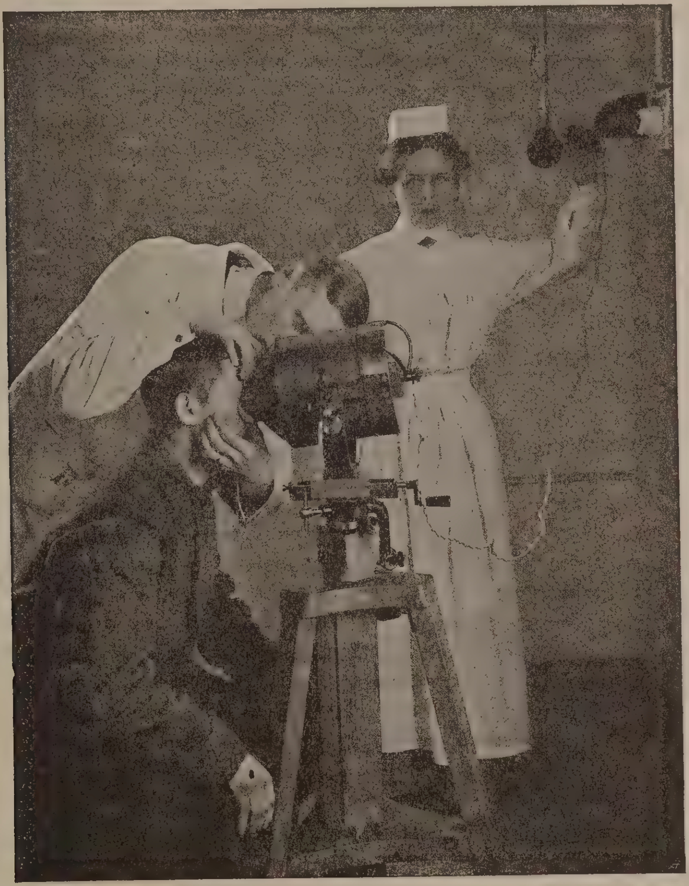


FIG. 79.—Magnet Extraction.

III. Magnet Extraction

The application of the electromagnet serves not only to diagnose the presence of a spicule of iron in the eye, but also at the same time to remove it. The credit for having made this instrument useful in ophthalmology belongs to *Hirschberg*. *Hirschberg's* hand magnet serves to extract the spicule from either the vitreous, or the anterior chamber, after an opening has been made in the capsule of the globe. The giant magnet was devised by *Haab* and has been modified in various ways. A number of different models are now in use.

We always begin with the giant magnet. After we have cleansed the neighborhood of the eye and the conjunctival sac, and have instilled cocain, we bring the eye carefully toward the instrument. If the lens has been injured, as in this case, we cause the magnet to act through the centre of the cornea. The patient feels a sharp pain as his eye approaches it, and, behold, here is the spicule on the magnet, ready to be weighed. It has returned through the lens and has passed through the wound of entrance in the cornea. We need only to bandage the eye and, later, to remove the traumatic cataract by operation. When the lens is uninjured we try to draw the spicule of iron through the pupil into the anterior chamber. To do this we apply the magnet toward the peripheral part of the iris and interrupt the current as soon as the foreign body appears behind the latter, in order that the tissue may not be perforated. Then we apply the magnet in another place so as to try to guide the foreign body through the pupil into the anterior chamber. After this has been done we need *Hirschberg's* hand magnet. The anterior chamber is opened with a keratome, the point of the magnet is introduced and then the spicule is easily extracted, as a rule.

There are cases in which the use of the giant magnet is contraindicated, as when the splinter of iron has been in the eye for a long time, or is firmly enveloped by a connective tissue capsule. In such a case we try to locate it by means of the sideroscope, open the sclera by a meridional incision, and introduce the point of the hand magnet directly into the eye. The introduction of the magnet was a great advance in ophthalmic therapy; by means of it we can save a portion of the vision of a great many eyes which otherwise would be lost. When the lens is not injured it is possible for perfect vision to be regained.

96. Prophylaxis of Injuries of the Eyes and of Their Complications

No one can fail to perceive that injuries of so valuable an organ as the eye enter deeply and in many ways into the questions of the existence of the individual, the welfare of the family and social economics. The importance of these injuries is so great that it can be readily understood why much has been done to prevent them by means of an efficient prophylaxis. Many safety devices have been adopted in the individual trades, and I strongly advise that every opportunity be taken to study them. Protective screens are to be found in the metal and iron industries to intercept any foreign bodies that may fly off, and suction contrivances may be seen attached to machines. Naturally we cannot deal with all of these things, but shall consider only the prophylaxis of injuries of the eyes in so far as it pertains to medical activity.

First a word concerning protective spectacles. Many artisans, if not the majority, are outspoken in their objections to the wearing of them. Anyone who will wear a pair for a while can readily convince himself that they possess certain inconveniences and disadvantages. Many of them interfere with the vision. They cannot but become dirty during work, they become scratched, tarnished and dull. Moreover, they increase the trouble of an artisan who has an error of refraction, or whose vision is otherwise impaired. Other workmen complain of a feeling of heat in the eyes, especially those who wear the closed models. To appreciate this feeling it is only necessary to wear for a while a similar pair of automobile goggles. The perspiration accumulates behind such spectacles. When the work is about a fire the metal frames may become quite hot. We also find that those who wear protective spectacles often suffer from a chronic hyperæmia of the conjunctiva and tend to have more severe conjunctival catarrhs. Protective spectacles that have been worn a long time are often dull with dirt, and then they form direct sources of danger of infection to the environment of the eye, and even to the eye itself. Experiments performed in the clinic of *v. Michel* have proved that infectious germs may lodge on the cilia directly from such infected spectacles. Other protective spectacles are too heavy, and cause the workmen to complain of headache, pain at the root of the nose and in the ears. One of the most serious objections is that many models do not furnish satisfactory protection against injury. The breaking of the glasses themselves have given rise to injuries of the eye, and, on the other hand, models have been used which from their construction were unable to prevent injuries.

These unquestionable inconveniences and disadvantages do not render the wearing of protective spectacles unnecessary in the various trades. On the contrary every physician should impress, by advice and warning, that this is one of the most efficient measures that can be taken in the prophylaxis of injuries of the eyes. Still it is necessary that these inconveniences be ameliorated and that every effort should be made to improve the models. It is not every form of spectacles that is suited for a particular trade. We have here: 1. Protective spectacles suitable for workers in hard materials who need accurate vision. Here again the workmen require different models according as the spicules of metal cast off during the work are very minute, or of a larger size. 2. Spectacles suitable for workmen in hard substances who do not need such sharp vision. 3. Spectacles for workmen in hot, or corrosive substances, a spattering, or a spilling of which is to be feared. 4. Spectacles for workmen exposed to glaring light. The injurious effect of the ultraviolet rays is particularly called to our attention by a number of models that are constructed with glasses that absorb these rays of light.

The physician can take part in various other ways in the prophylaxis of injuries of the eye. I believe that within the sphere of his influence the physician is an efficient educator who has ample opportunity to display his powers in contest with the indolence, stupidity and ignorance of the populace. Children furnish a very large share of the gravest perforating injuries of the eye; hence, the old saw cannot be repeated too often in school and at home: knife, fork, scissors and fire are not fit for little children. Wherever opportunity offers the physician should promote by instruction and warning the prophylaxis of these forms of injury to the eye.

Another form of prophylaxis is that of the consequences of injuries of the eye. This consists of the careful observation of patients. Recall that superficial injuries of the cornea are very apt to lead to ulcus serpens in the people of the laboring classes, especially when the patient has a chronic dacryocystitis. *Axenfeld* is right in his claim that the prophylactic extirpation of the diseased lacrymal sac is one of the most important measures that can be taken to prevent ulcus serpens. The timely advice of the physician can prevent many a misfortune. Even when the injury has occurred, he can do much in the way of prophylaxis. I do not mean by this that he can undo the injury, but that he must try in every way to mitigate its consequences and to prevent complications. I will make myself clear with a few practical examples.

If an ulcus serpens is to be feared, or if one is already in progress, after a superficial injury of the cornea, the patient should receive a subcutaneous prophylactic injection of pneumococcus serum and have his eye dressed; then the aid of the specialist should be sought, for extirpation of the lacrymal sac and for the further treatment of the ulcer.

Foreign bodies in the conjunctival sac, and especially in the cornea, should be removed as quickly as possible by a physician. The workman very often seeks to have such foreign bodies removed by his comrades, but unskillful hands and dirty instruments frequently give rise to great trouble.

If there is any suspicion that an eye has been injured by a penetrating splinter of iron the patient should be taken to the ophthalmologist at once that he may try to remove it with a magnet as quickly as possible.

Haste is not usually so necessarv in contusions of the eye without rupture of the globe, but a case in which the lens has been dislocated into the anterior chamber forms an important exception. The immediate removal of the lens is imperative in order to prevent the rapid development of a secondary glaucoma. In perforating wounds the physician first consulted should cleanse the eye and the wound and then apply an aseptic dressing, in order to do everything possible to prevent infection. If the wound is already infected we have to take into consideration the safety of the second eye, as well as to care for the injured one. Desirable and necessary as it is that injured eyes should receive the aid of the ophthalmologist as quickly as possible, yet the general practitioner can do a great deal in the way of prophylaxis.

97. Cranial Injuries and the Eye

We have to examine a patient who fell three months ago and fractured the base of his skull. His statement that one eye became blind immediately after the injury has been doubted. The eye may be involved in many ways, and the changes met with in that organ are diagnostically valuable in injuries of the skull. When the eye is evidently involved in such an injury the course of the examination seems to guide itself. We first notice what injuries have been suffered by the soft parts about the eye, then examine into the condition of the walls of the orbit and finally ascertain what injuries have been inflicted on the eye itself. These injuries present the most manifold combinations of lesions, but on the whole they may be classed into six predominant types: 1, Hemorrhage into the orbit and exophthalmos; 2, emphysema of the orbit; 3, fractures of the bones of the orbit in connection with fractures of the base of the skull; 4, lesions of the optic nerve in connection with fractures of the base of the skull; 5, lesions of the central optic tract; 6, pareses of the ocular muscles.

I. Orbital Hemorrhages and Exophthalmos

When a severe contusion falls upon the bones of the face and skull in the vicinity of the eye, i.e., upon the frontal, malar, superior maxillary and nasal bones, or upon the bony framework of the orbit, a hemorrhage takes place into the cavity in the majority of cases. An orbital hemorrhage is in fact the commonest symptom, and the first one we look for in such injuries of the skull. The hemorrhage may come from lacerated vessels in the orbit, or from fractures in the wall of the orbit and, through these, from the accessory sinuses. The blood may force its way into the orbital fat, into *Tenon's* capsule, into the vaginal space of the optic nerve, or beneath the periosteum of the bones.

The principal symptoms of an orbital hemorrhage are exophthalmos and sugillations of the conjunctiva and lids. The skin escapes discoloration only when the blood is confined beneath the periosteum. The exophthalmos can be perceived at once; the eyeball is displaced forward, laterally, or upward, and its movements are restricted in accordance with the position and extent of the hemorrhage. The limitation of movement is due to the contraction of the space in the orbit, as well as to the compression of the nerves and muscles. It may be mentioned in this connection that the hemorrhages that follow contusions may be very great in persons who are hæmophilic, and that in such persons the hemorrhages may even occur spontaneously from stooping, or coughing.

We have to consider what effect may be produced by an orbital hemorrhage when it is the only symptom present on the part of the eye in an injury of the skull. The exophthalmos may be so great as to prevent the closure of the lids, and then the cornea is exposed to the danger of becoming dry. Mydriasis, due to pressure on the nerves, is observed not infrequently. Above all, the vision may be impaired, and this impairment may increase to blindness, when the orbital hemorrhage is very great, because of the pressure exerted on the optic nerve and on the blood vessels that supply the eye, but an amaurosis that appears immediately after an orbital hemorrhage indicates a direct lesion of the optic nerve almost with certainty, even though no changes can be recognized at once with the ophthalmoscope, for such a hemorrhage alone cannot make the optic nerve totally incapable of performing its functions. Although a hemorrhage into the orbit may occur after traumatism to the head without fracture of its bones, yet in many cases, as *v. Bergmann* in particular has insisted, the existence of a fracture of the base of the skull may be diagnosed from its occurrence.

The treatment of an orbital hemorrhage depends on the case. Generally the blood undergoes spontaneous resorption, and it is only in cases of subperiosteal hæmatoma that the process is very much prolonged. In such cases we content ourselves with a pressure bandage. When the exophthalmos is extreme, the injury fresh, and the lagophthalmos endangers the eye, incisions, or punctures, may be made to evacuate the blood. In one such case I withdrew 15 ccm of blood by means of a syringe and obviated the exophthalmos.

II. Emphysema of the Orbit

Exophthalmos is the first symptom we notice in an orbital hemorrhage. When we try to press the eyeball back we feel the resistance that is opposed to our efforts by the orbital tissues that are filled with blood, but sometimes the feeling is different. Air, instead of blood, occasionally enters the orbit, or the eyelids, in injuries of the skull, and then we have an emphysema.

We may differentiate three varieties of emphysema of the eye: the purely palpebral, the orbitopalpebral and the orbital. The purely palpebral is very rare, but is met with occasionally in a fracture of the lacrymal, or nasal bone. The orbital, or orbitopalpebral, is much more common. Air enters the orbit after fracture of the lamina papyracea; if it accumulates behind the septum orbitale an orbital emphysema is produced, but if part of it penetrates beneath the skin of the lid we find the orbitopalpebral form. This differentiation has a certain clinical importance. The lids are swollen in all three forms. In palpebral and orbitopalpebral emphysema we can feel the crepitation of the air beneath the skin, but the eyeball is not protruded, while in orbital emphysema there is always a great exophthalmos together with the swelling of the lids. The latter form is therefore of greater interest to us in injuries of the orbit.

Orbital emphysema is easily recognized: the eyeball protrudes, the lids are swollen, the palpebral fissure is closed. The protrusion of the eyeball is associated with limitation of motion and diplopia. The swelling of the lids differs from that in palpebral emphysema in that it extends only to the margin of the orbit and remains at a distance from the margin of the lid. The skin of the lid is pale. When we press the eye back the resistance offered is not as great as in orbital hemorrhage and no crepitation is felt beneath the fingers. The swelling gives a tympanic note on percussion.

The exophthalmos that may thus be produced in an orbital emphysema has an even greater signification in the diagnosis of a cranial injury than one due to an orbital hemorrhage. *An emphysema always tells us that there is a fracture of the wall of the orbit*, for it can occur only when a communication has been formed between the orbit and one of the accessory sinuses that contains air. Most often it is the inner wall of the orbit, the lamina papyracea of the ethmoid, that is fractured, but, more rarely, the fracture may extend into the frontal sinus, or the antrum of *Highmore*. According to the researches of *Heerfordt* the orbit in such a case is under a mercurial pressure of 80 mm, the highest pressure in man of physiological expiration. The explanation of the origin of an emphysema is that during an expiratory increase of air pressure, caused usually by blowing the nose, the air is forced out of the accessory sinus into the orbital tissues. The patient frequently feels a darting pain, but the time after the injury at which the emphysema appears varies a good deal. Usually the patients are impelled to blow the nose on account of bleeding, so in most cases it appears shortly after the injury, but sometimes it is delayed for several weeks.

Little is known as yet of the way in which the orbital wall becomes fractured in cases of orbital emphysema. *Fuchs* is of the opinion that when the eyeball is struck at the time of the injury it is forced backward against the orbital fat, which yields in the same manner, and crushes the delicate lamina papyracea. *Salus*, on the other hand, was able to produce fractures in the lamina papyracea in the cadaver by blows upon the margin of the orbit. It may be mentioned in this connection that there are persons who are accustomed to take snuff in whom an emphysema may be produced spontaneously on blowing the nose without any preceding traumatism, and in whom this may occur repeatedly. It is probable that in such cases the ethmoid cells are injured to such an extent by the habitual increase of the air pressure that they become broken inward toward the orbit.

Although an emphysema of the orbit may appear very serious at first glance, its course is usually benign. The only treatment necessary is to apply a pressure bandage and to forbid any action that may tend to cause increased air pressure, particularly any blowing of the nose. The air will then become resorbed in a few days. It is only when the exophthalmos is extreme that the air may be evacuated by an incision.

III. Fractures of the Bones of the Orbit and Enophthalmos Traumaticus

When the region of the eye is involved in an injury of the skull we must not content ourselves with palpation of the soft parts and of the eyelids, no matter whether it is a hemorrhage, or an emphysema that renders a fracture of the wall of the orbit probable; on the contrary we must try to determine whether the fracture of the wall is direct or indirect.

Direct fractures of the bones of the orbit are those that take place at the immediate point of impact, indirect fractures take place elsewhere, but this differentiation is not always easy. Ordinarily a direct fracture is associated with a lacerated, or contused, wound of the skin and often can be felt. A point of greater tenderness may indicate the place of fracture and, if the fragments are movable, crepitation may be detected. Indirect fractures can neither be seen nor felt, and the skin over them is intact, because the blow fell on another place. Very small fissures may even evade observation in X ray pictures, so we often have to depend on the accompanying symptoms to guide us to a diagnosis of an indirect fracture of the orbit. Symptoms more or less common to all fractures of the walls of the orbit are orbital hemorrhages, displacement of the eyeball, especially exophthalmos, emphysema and hemorrhages from the accessory sinuses, but we must note above all that the eye itself, or the optic nerve, is quite apt to be injured. The eye may be destroyed and even torn out, while the optic nerve may be crushed, or compressed by hemorrhages, and so give rise to visual disturbances.

In certain forms of accident, in which the head is compressed, the eyeball may be squeezed out, or torn out of the orbit. We speak of luxation of the globe when the eyeball lies in front of the orbital septum with the muscles and the optic nerve tensely stretched, and the lids closed spasmodically behind it; of avulsion when the globe remains attached only by some shreds of tissue. Such injuries occur when the bony walls of the orbit are pressed together, as the fracture takes place, and the eyeball is pried, or pressed out. Luxation and avulsion of the eyeball are met with not only in serious accidents, but also during labor, in the course of a forceps delivery.

It may be mentioned in this connection that lunatics sometimes attempt to mutilate themselves by luxating the eyeball and occasionally tear it out completely. They accomplish this by inserting a finger between the margin of the orbit and the eyeball so as to pry out the latter. Sometimes they try to tear through the muscles with the fingernails.

Thus we have a series of indications when a fracture of the bones of the orbit is present and we try to determine from them the situation of the fracture. It is of less interest to us to differentiate between fractures of the margin and of the wall of the orbit, especially as fractures of the margin are usually combined with fractures of the wall, than to determine whether the fracture is direct or indirect.

Fractures of the upper wall of the orbit are met with in association with those of the frontal bone and of the base of the skull. Fractures of the middle and posterior fossæ are apt to extend to the anterior fossa. The supraorbital nerve may be injured in fractures of the upper margin of the orbit and fissures of the roof of the orbit frequently extend into the foramen opticum. Fractures of the lower wall of the orbit occur chiefly in connection with injuries of the malar and superior maxillary bones. In such cases a hemorrhage takes place into the antrum of *Highmore*, blood escapes from the mouth and nose, and injuries of the infraorbital nerve, the rectus inferior and obliquus inferior may be observed. In many cases the eyeball is displaced into the maxillary antrum. It should be noted particularly that the lacrymal passages may be injured in fractures of the lower margin of the orbit.

In fractures of the inner wall of the orbit, which are produced sometimes by the entrance of foreign bodies at the inner canthus, sometimes in connection with fractures of the base of the skull, we meet with hemorrhage from the nose and emphysema of the orbit, and very often find the internal rectus injured. Fractures of the outer wall of the orbit occur principally when the blow falls on the temporal and the frontal processes of the malar bone. The chief symptom is exophthalmos with hemorrhage from the mouth and nose.

Enophthalmos Traumaticus

A peculiar clinical picture, characterized by a sinking backward of the eyeball into the orbit, is not infrequently met with in connection with these injuries. This condition is known as enophthalmos traumaticus. It occurs after a violent contusion on the bony margin of the orbit, or its immediate environment, as when the blow falls on the nose, forehead, malar bone, cheek, or in many cases on the side of the head. In every one of the cases I have thus far seen the condition was produced by the kick of a horse.

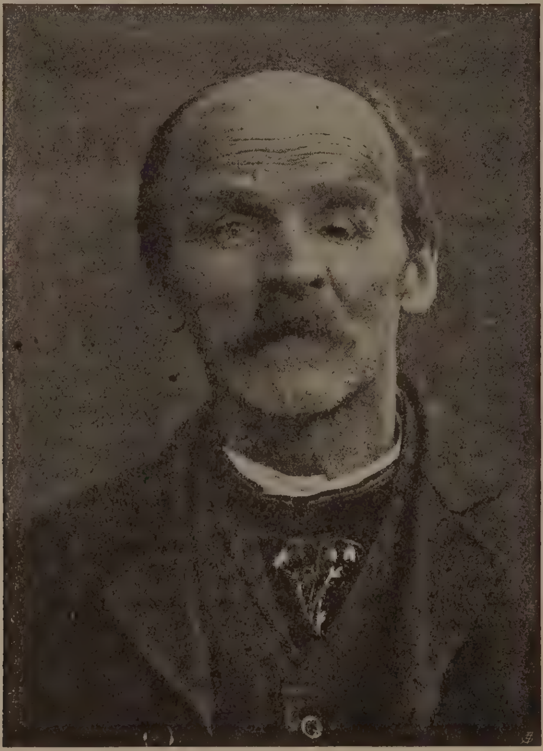


FIG. 80.—Traumatic Enophthalmos of the Left Eye after a Kick of a Horse.

A glance at a patient with enophthalmos traumaticus gives the impression, as *Nieden* has aptly said, that he is wearing an artificial eye (see illustration). The palpebral fissure is narrowed, the lids are sunken and the palpebral fold of the upper lid is drawn deeply beneath the margin of the orbit. The mobility of the lids is usually limited. Two important features are that the eyeball has receded into the orbit and that the margin of the latter is usually deformed with scars in the surrounding skin. The eyeball itself may be normal as regards its internal condition and its mobility, but its vision is often reduced, even to blindness, and in the majority of cases its mobility is limited in one or more directions. The clinical pictures vary a great deal. The reason for this will be readily understood when we come to study the pathogenesis.

Several factors, which may combine, take part in the production of this anomaly. In one case the principal factor is an enlargement of the space in the orbit, caused by the separation of the fragments of the broken bones, so that the eyeball seems to be disproportionately small. In another case the contents of the orbit become reduced in volume simultaneously with the deformation of its neighboring tissues. A large portion of the orbital fat may be displaced through a gaping wound into one of the accessory sinuses and this tissue may become atrophic to a great extent, through a fibrous condensation and shrinkage as the result of hemorrhages and processes of organization. Not rarely the case is one of pressure necrosis and pressure atrophy. In this way the eyeball is caused to recede.

It was formerly thought that this contraction of the orbital tissues was a sort of trophoneurotic atrophy, caused by an injury to the trophic nerve fibres in the sympathetic and trigeminus, but this theory has been abandoned. The eyeball is drawn directly backward by the fibrous contraction of the contents of the orbit. Extensive fractures are always associated with great cicatrization.

It has been correctly pointed out that the fascia, which primarily guarantees the fixation of the eyeball, may also be injured in fractures of the orbit. The enophthalmos appears sooner or later after the injury, as one or another of these factors predominates, but ordinarily a period of several weeks is necessary for its development.

Therapeutically we are impotent in cases of traumatic enophthalmos. The deformity is permanent. If the patient is troubled with diplopia the eye must be covered so as to exclude it from the act of vision.

After we have examined the margin of the orbit we investigate the lacrymal passages, for ruptures of the canaliculi, of the mucous membrane of the lacrymal sac, or of the entire lacrymal sac, are apt to be produced by contusions in this region. After a kick of a horse that has broken the bony framework of the nose we not infrequently find an injury of the lacrymal canal which gives rise to a dacryocystitis.

IV. Lesions of the Optic Nerve in Fractures of the Skull

We come now to the important question, whether the optic nerve has been injured. The involvement of the optic nerve is a frequent complication of injuries of the skull. Under certain circumstances it may be compressed by an orbital hemorrhage alone, but it is of diagnostic value to know that generally speaking a simple orbital hemorrhage is of no great importance in the production of permanent lesions of the optic nerve, because the pressure can scarcely make this thick nerve incapable of conduction. At the most the optic nerve may be stretched, or a transient circulatory disturbance may be produced in its vessels by the pressure. When a permanent impairment of vision follows an injury to the skull, as in this patient, it has some other cause. Two things are to be considered from an anatomical point of view.

Lesions of the Optic Nerve at the Foramen Opticum and Hemorrhages into Its Sheath

First of all there may be a direct injury to the optic nerve at the foramen opticum. We know from numerous observations that fractures of the base of the skull are very apt to pass through the optic foramen. Hence the optic nerve may be torn across, or at least crushed and compressed, very easily at this place by the displacement of fragments of bone, or by the breaking off of splinters from the lesser wing of the sphenoid. This fracture in the optic canal may be met with in all forms of injury to the skull, but it usually occurs when the frontal bone and the margin of the orbit is affected. When the impact of the blow falls on the opposite side of the head the fracture in the orbit is indirect.

In cases of fracture of the base of the skull in which the patients do not lose consciousness and are able to observe their symptoms, they may themselves remark a blindness of one eye immediately after the injury. If we examine such an eye with the ophthalmoscope immediately after the injury it appears to be normal, but after the lapse of some time, usually two or three weeks, a descending atrophy develops in the optic nerve. We can see the atrophy in the eye of this patient and can feel perfectly certain that the conductivity of the nerve has been broken in the optic canal. In some cases we find at first only a slight hyperæmia, or signs of engorgement in the optic nerve.

The behavior of the pupil is of much greater importance in such a case when we have to make an early examination. If the optic nerve has been torn across direct reaction of the pupil of the injured eye and consensual reaction of the other pupil cannot be excited by light, while a consensual reaction must be obtained when light is thrown into the healthy eye. The amaurosis is immediate and permanent in the overwhelming majority of these cases.

Sometimes it happens that a circumscribed defect in the field of vision appears immediately and remains permanently after an injury, and that, later, a partial, white, atrophic discoloration of the papilla can be perceived. In such a case the diagnosis is a partial laceration of the optic nerve in the optic canal.

Exceptional cases are met with in which the vision is greatly impaired at first and then gradually becomes restored. In these cases the nerve was temporarily compressed at this place.

When there is a great impairment of vision, or blindness of both eyes after a fracture of the base of the skull, there may be a bilateral fracture in the foramina optica, or a fracture of the sella turcica. Clinical experience has shown that when such a patient continues to live he does not suffer from bilateral amaurosis, but that a portion of his vision is restored. Indeed, cases of complete restoration of vision have been reported.

Hemorrhages into the Sheath of the Optic Nerve

Hemorrhages take place into the sheath in almost all cases in which the optic nerve is wounded at the optic canal in fractures of the base of the skull, and, doubtless, a part of the blood comes from the lacerated vessels in the canalis opticus. Intracranial effusions of blood may also enter the sheath when there is no fracture in the optic canal, for example, when the middle meningeal artery is ruptured. The accumulation of blood in the subarachnoid and subdural spaces of the sheath of the optic nerve may be so considerable as to cause an ectasia of the sheath; this condition is called a *hæmatoma of the sheath of the optic nerve.*

The diagnostic value of these hemorrhages into the sheath of the optic nerve was formerly much overestimated, an echo of which may still be heard when the diagnosis is well marked. *Knapp, Berlin* and others believe that such a hemorrhage may compress the central vessels of the retina and induce sudden blindness with the clinical picture of an embolism of the central artery, but we know from the findings in numerous autopsies that the ophthalmoscopic condition is perfectly normal in the subdural hemorrhages into the optic nerve sheath, which are so frequently slight, that are met with in fractures in the optic canal. We also know that the fundus of the eye may be perfectly normal even in extensive hemorrhages into the optic nerve sheath. It is also true that we may meet with a hyperæmia of the papilla after a fracture of the base of the skull when there is no hemorrhage into the sheath of the optic nerve. It is only in subarachnoid hemorrhages, which may enter the sheath of the optic nerve from the cranial cavity, that we sometimes find a papillitis shortly after the injury. Hence, a hemorrhage into the sheath of the optic nerve can be diagnosticated with the ophthalmoscope only when a papillitis is present. Everything else is conjecture. A passage of the blood from the optic nerve sheath through the lamina cribrosa has not been proved, even in the rare cases in which a black pigmentation of the scleral ring and of the atrophic papilla has been observed at a later period. It is probable that in such cases hemorrhages have taken place from the vessels of the papilla and retina.

In addition to these primary changes, secondary ones may appear in the optic nerve after an injury to the skull. A papillitis may be produced by the increase of intracranial pressure occasioned by hemorrhage; this is to be seen usually a short time after the injury. We also meet with a neuritis in the post-traumatic, hemorrhagic softening of the brain, as well as when a meningitis supervenes upon an injury of the skull. Finally, it happens not so very rarely that a tumor cerebri develops years after a traumatism to the skull and causes a choked disc.

V. Lesion of the Central Optic Tract after Injuries of the Skull

All portions of the course of the optic fibres may be affected in cases of cranial injury. The chiasm, the optic tracts, the intracerebral bands and the cortical visual centre may all be harmed in the greatest variety of ways. I can give only a few diagnostic points. When the chiasm is injured we find either bitemporal hemianopsia, or amaurosis of one eye with temporal hemianopsia of the other; when the blindness is bilateral and the fracture is located in the anterior portion of the base of the skull we are unable to say whether the chiasm has been completely destroyed, or both nerves have been lacerated somewhere along the base of the skull, or in the canalis opticus. All lesions above the chiasm must produce homonymous hemianopsia. Lesions of the cortical visual centre have been observed to be particularly frequent in injuries of the skull. When the right visual centre is injured the hemianopsia is on the left side. When the destruction of the centre is incomplete hallucinations and subjective sensations of light sometimes are produced. In uncomplicated cases the condition seen with the ophthalmoscope is perfectly normal. It is a matter of great interest to know that when a concentric contraction of the remaining portions of the field is met with in connection with traumatic homonymous hemianopsia, a combination of traumatic neurosis and hysteria must be suspected.

VI. Lesion of the Nerves of the Ocular Muscles in Injuries of the Skull

After we have taken into account the most important clinical types in the examination of a cranial injury with involvement of the eye and its surroundings in the way just described, after we have ascertained whether an orbital hemorrhage, an emphysema, a fracture of the orbit, or a lesion of the optic nerve, or of the optic tract can be demonstrated, there still remains one thing more for us to do, we must determine whether any of the ocular muscles are paralyzed.

The abducens is the most frequently affected of the nerves that supply the ocular muscles. In fractures of the base of the skull it is very apt to be lacerated at the place where the apex of the petrous portion of the temporal bone becomes horizontal. The facial nerve also is frequently injured in fractures of the skull, either alone, or together with the abducens. In most cases the paralysis is unilateral; bilateral paralysis occurs only in grave transverse fractures.

It may be mentioned in this connection that paralysis of the facial nerve is met with very frequently after labors in which the child has been delivered with forceps, but this paralysis is peripheral, produced by compression of the nerve after its exit from the stylomastoid foramen. If a facial paralysis appears at a later period as secondary to a fracture of the base of the skull, an inflammation is to be suspected. The cases of traumatic facial paralysis have also furnished evidence that the secretory nerve of the lacrymal gland comes from the facial.

The oculomotorius is paralyzed in injuries of the skull much more rarely than the abducens and the facialis. In such a paralysis we must think primarily of a basal injury. In many cases the lesion may be situated in the fissura orbitalis superior, when a fracture extends into the roof of the orbit and also involves the optic canal. When the oculomotorius and abducens are simultaneously paralyzed there may be a fracture of the petrous portion of the temporal bone, because these two nerves lie close together at this place. If the oculomotorius is paralyzed at the same time as the opposite side of the body, the lesion is in the peduncle. The trochlearis and trigeminus are affected least often of all. When they are paralyzed the basal site is likewise the first to be considered. In all of these traumatic paralyses we have to consider whether, in any given case, the probability is in favor of an orbital, a basal, a nuclear, or a cortical lesion.

98. Concentric Contraction of the Field of Vision in Traumatic Neuroses

I am about to introduce a patient who is engaged in a lawsuit against a railroad, in the course of which he has thus far been examined by more than twenty physicians. He received a trivial injury in a railroad accident and has developed a clinical picture which must be considered one of traumatic hysteria. I wish to present the case because it gives me an opportunity to demonstrate important ocular symptoms that are to be met with in neuroses that are induced by accidents. I will quote a few symptoms from his voluminous clinical history.

No organic change is to be found in the patient. In walking he stands on the toes of his right foot, after sliding the sole of the foot slowly, but completely along the floor, and then the left leg hangs like a dead weight, extended at the knee joint, forming a right angle at the ankle joint, while the left side of the pelvis is lifted at the same time. The left foot can then be seen and heard to slide along the floor. If he stands on his right leg the left is lifted from the floor only a little and for short intervals. He is unable to obey when directed to stand on his left foot. The left arm for the most part hangs down like a dead mass and is almost always moved passively with the right hand. This happens even when the movement to be executed could be accomplished by gravity alone. The left thigh is considerably smaller than the right. The sensitiveness of the entire left side of the body, with the exception of the left side of the nose, is greatly impaired. On the left side of the body gentle touches are not felt, pricks are not painful, but are felt as touches and correctly located; a sharp instrument cannot be distinguished from a blunt one, neither can heat be distinguished from cold. As regards the reflexes, the left corneal reflex is the weaker, as is also the left pharyngeal reflex. The tendon reflexes are all present and both *Babinski's* symptom and *Struempell's* toe phenomenon are absent. The plantar reflex cannot be obtained on the left side. He complains of paralysis of his left arm and leg and of a constant itching of his right arm and leg, which sometimes augments and becomes a violent pain. His gait is uncertain, he has constant headache, he has a feeling of wretchedness, of weariness of the battle of life, and prefers to shut himself up in a dark room. Heart troubles particularly annoy him. He has lost the power of localization, he is very forgetful, he is very easily vexed, his tone very quickly becomes whining. He no longer speaks fluently, but stammers and stutters, although his intelligence is unimpaired. These are only a few of the important points to be gleaned from this thick bundle of legal documents. The thorough examination made by the neurologists has proved that no organic disease is present, so there can be no doubt that this is a case of traumatic hysteria, the actuating cause of which was the railroad accident. The patient, who may repeat the history of his accident, now complains of a number of troubles referred to his eyes. It may be noticed that while I have been speaking a tic has appeared on the left side of his face, especially about the eyes. When we come to examine the eyes we find the disturbance to be purely functional, the pupillary reactions and the ophthalmoscopic conditions to be normal. Here is the reason I present this case: Both visual fields are very much contracted concentrically. Patients in whom the visual field is contracted to such a degree in consequence of retinal diseases are scarcely able to go about alone, but it is characteristic of this functional contraction of the field that the orientation does not suffer. This concentric contraction of the visual field is the commonest functional disturbance of the eye. It may be unilateral, or bilateral.

It is also of diagnostic interest to know that there are certain relations between the concentric contraction of the visual field and the disturbances in the sensibility of the skin, especially in hysteria. As a rule, it is met with in those cases of hysteria that have cutaneous anæsthesias, such as are present in this patient. The question arises whether these symptoms are not those of aggravation and simulation, and in fact, it is not always easy to distinguish between an exaggeration and the existence of a neurosis caused by an accident, but there is no doubt that this concentric contraction of the visual field is a frequent occurrence in traumatic neuroses. In such cases we test the visual field at different distances. Under certain circumstances we are able to determine, by following the directions given by *Schmidt-Rimpler*, whether the contraction is simulated. If the visual field remains of the same size at different distances and the vision of the patient is good, the suspicion of aggravation naturally arises, but we must be careful about such suspicions in hysteria, for the suggestibility of the patient must certainly be taken into account.

Another form of disturbance of the visual field that is met with in traumatic neuroses is *Foerster's* displacement type. This phenomenon depends on the fact that the appearance of an object on the perimeter exerts a greater stimulation than its disappearance. For example, if we move the object on the perimeter from the temporal side toward the centre, the patient perceives it at 90°, and then, if it is moved slowly beyond the centre, it disappears at 30° on the nasal side, while it should be seen as far as 50° or 60°. If we then reverse the procedure and start in the nasal portion of the field the object is perceived at 50°, but disappears on the temporal side at 60°, although in the first test it was seen at 90°. The importance of thus testing the visual field in all cases of traumatic neurosis is considerable, though it should not be overestimated. The behavior of the visual field should be taken into account before passing judgment on such cases. Occasionally we meet with other ocular symptoms besides the concentric contraction of the visual field in cases of traumatic neurosis. The patients frequently complain of great photophobia, dazzling, lacrymation and blepharospasm, as well as of light phenomena, such as fluttering, fiery balls and muscæ volitantes. Many forms of visual disturbance may also be present without any objective condition to furnish an explanation. Thus objects may gradually disappear, weariness may come on rapidly, or there may be amblyopia, which may even increase to hysterical amaurosis. An amblyopia or amaurosis is usually associated with a hemianæsthesia of the same side. In this connection it may be mentioned that a transient concentric contraction of the visual field with impairment of the central vision, may be observed in many epileptics immediately after a fit. Marked symptoms of paresis and contracture of the ocular muscles may also be observed in hysteria. These neuroses due to accidents may occur after injuries of the body, as well as after slight, or severe injuries of the eyes.

99. Introduction to the Judicial Medical Opinion in Injuries of the Eye

Every physician in Germany is likely to be called upon to form a judicial opinion on the earning power of a workman whose eye has been injured. I admit that an eye clinic is not the proper place in which to study social legislation and the details of accident insurance, but, as I am well aware of the helplessness a physician is apt to feel when he is required to form an estimate of the compensation due the victim of an accident, I am accustomed to give my students some advice and practical suggestions in regard to this matter and to reason out with them the proper indemnity in such suitable cases as we happen to meet with. A few introductory remarks may not be out of place.

As death is practically not to be taken into account, because other lesions are met with in fatal injuries, the purpose of the accident insurance law, in so far as it affects workmen whose eyes have been injured, may be thus stated:

The expenses of treatment shall be borne from the beginning of the fourteenth week, and an income shall be furnished the injured person for the period of his incapacity to work. This income is 66⅔% of the yearly wage for total disability, or a fraction of that amount for partial disability.

Every accident must be reported by the man in charge of the work to the local police board, stating the nature of the accident, the residence of the person injured, etc. The executive organs of the accident insurance law are the mutual trade associations (Berufsgenossenschaften). Appeal from the decision of the mutual trade association may be made to the arbitration board (Schiedsgericht), and from the latter to the Imperial Insurance Office (Reichsversicherungsamt).

The duty of the physician in the carrying out of the accident law is twofold: both medical and medicolegal. If he is to be efficient he must have an accurate knowledge of the accident insurance law. He must know what an occupational accident is, and what disability, or loss of earning power, is in the meaning of the law. He must of course oversee and govern the treatment, and must, finally, be able to draft a professional opinion.

An ophthalmological opinion is constructed systematically, the same as any other. It contains: 1, an introduction concerning its motive and purpose; 2, a short recapitulation of the treatment thus far, or a review of the accident based on the accompanying legal papers; 3, the subjective statement and disorders of the person injured, or the report of his relatives; 4, the objective conditions found on examination; 5, the diagnosis; 6, the opinion proper concerning the connection of the clinical symptoms with the accident and concerning the effect of the consequences of the accident upon the earning power; 7, the prognosis. This entire apparatus of the accident insurance law, from the first report of the injury to the final act, is to serve a great, noble and humane purpose—the accident indemnity.

The purpose of our legislation is that the loss of earning power, which exists from the beginning of the fourteenth week after an accident, shall be compensated for by an income. The injured person receives this indemnity not for the injury as such, but only for the diminution of his earning power occasioned by an accident incurred in obtaining his livelihood. When a workman suffers an injury to his eye which partially impairs his earning power, this loss of earning power has to be estimated. By whom is this estimate to be made? It would seem at first as though this should be the duty of the employer, or of an expert in the trade, for one would think he would be able to decide most surely what the injured person can do and what he cannot. Such a tribunal should also have a medical certificate giving an accurate statement of the pathological changes that have taken place. But the laity are not always able to determine what degree of earning power is present, even with the aid of an excellent medical certificate. The development of the practice of accident insurance proved this very quickly.

It is not the expert, or the judicial court, that is called upon first for an estimate of the degree of loss of earning power, but the physician. In his formal opinion the physician should give not only an accurate description of the morbid changes, but also an estimate in percentage of the loss of earning power suffered by the person injured. He does not make the award, he is not the final authority, but the award is based on his estimate. It may be questioned whether a physician is in a position to be able to judge the earning power a man retains in his particular calling after his eye has been injured, and this question has called forth very different answers. Some ophthalmologists maintain that a physician is absolutely unable to estimate the influence a bodily injury exerts upon the earning power. It must be admitted that, in view of the immense complexity of the affairs of life and the great multiplicity of vocations, he is often unable to obtain any proper conception of the conditions under which this or that work must be performed. It is impossible that he should know all the details of all departments of labor.

We, as ophthalmologists, can only determine primarily the working capacity of the eye through a comparison of its functions with those of a normal one; but the comparison of the working capacity of the injured eye with the normal gives no definite information in regard to the earning power of the injured person. Many factors enter into the very complicated conception of the earning power, a few of which are: 1, the demands made by the various forms of labor upon the eye; 2, the age of the person injured; 3, the estimate placed upon the working capacity of the employee by his employer; 4, the competitive ability of the workman; 5, the condition of the labor market. It is not an easy task for a physician to estimate the loss of earning power, and it can be readily understood why a large number of physicians wish that their estimates might be made optional. The diametrically opposite view is also held. Some ophthalmologists, and other medical scholars as well, look upon this duty as a very easy one. Ophthalmology possesses the most exact methods of examination. We can test the vision, determine the refraction, investigate the visual field and ascertain the binocular perception of depth; therefore it is believed that the ophthalmologist is the person best fitted to estimate the degree of the loss of earning power caused by the results of an accident, and that he can do it more easily and with greater certainty than anyone else. This opinion has been stated in the following words: "No branch of medical science can express in figures the occupational disturbance produced with greater accuracy than ophthalmology." (*Mooren.*) Mathematical formulæ have been devised for the purpose of making it possible to obtain an extremely accurate calculation of the accident indemnity that should be awarded in every ophthalmological case. Furthermore it has been proposed that ophthalmologists should systematically ascertain what degree of visual acuteness, of visual field and of stereoscopic vision is necessary for each particular form of labor and of occupation. Such a suggestion is practicable only to a slight extent, for, to carry it out, the physician himself must possess the technical skill to perform the most various kinds of labor.

As in many other things the middle road is the best. The physician cannot determine positively what portion of the earning power may be supposed to be still present with a certain ocular impairment, nor can the experts and mutual trade associations say with absolute certainty what earning power is still possible after the functional test has been ascertained. The two must work hand in hand. The duty of deciding concerning the award of an indemnity falls primarily upon the physician. Whether we accentuate the difficulties in the way of forming an opinion or not, the departmental boards rightfully demand from the physician a percentage estimate, for the physician is the first expert to testify. We can also comply with the demand, but we must always be conscious that we can make only approximate, not absolute, estimates.

We have at our disposal four methods by means of which to estimate in practice the earning power of a man whose eye has been injured.

1. *The Subjective Estimate*

Immediately after the enactment of the accident insurance law physicians were obliged to estimate the loss of earning power in a purely subjective manner, because no fixed data were to be obtained, and even in recent times, under the motto "individualization not schematization," many ophthalmologists have maintained that a greater scope should be allowed to subjective estimates. The injustice that has resulted, and still results, from such estimates is shown by this letter from a workman, in which it is stated that he lost an eye, that the physician who passed upon his case refused any indemnity because he continues to perform the same work in the factory as before, and that near him works a recently employed comrade who has likewise lost an eye, but receives $33\frac{1}{3}$ % indemnity. What an inflammable mass of anger and resentment may thus be accumulated!

It is perfectly true, as has been shown by statistics, that different workmen may have their earning powers impaired to very different degrees by similar accidental injuries. *Ammon* found fluctuations in this respect that ranged from 0 to 30% among persons having only one eye. Yet it cannot be emphasized too strongly that the victim of an accident should be made secure against the most unfavorable possibility of his condition. The physician cannot prophesy what consequences the accident may produce through changes in the conditions of labor. We cannot succeed without fixed standards and average values, and so we find that the subjective method is inadmissible in the formal appraisal of injuries of the eye. To administer justice we need fixed average values, so arranged that full credit may be given in each individual case. The question is how to find these standards and average values.

2. Calculation of the Earning Power by Mathematical Formulæ

A number of proposals have been made (*Zehender, Magnus, Heddæus, Berry, Becker, Szulislawski, Kazaurow*) to obtain a positive expression of the relation between an injury of the eye and the resultant loss of earning power by means of mathematical formulæ, but every attempt to do this is frustrated by the complexity of practical life. The impairment of the earning power of an injured person cannot be calculated by mathematics alone, the only estimate possible is approximate.

3. The Casuistical Method

It would seem as though the best way would be to collate a large amount of statistical material concerning the harm that really has resulted from accidents to the eyes. Many ophthalmologists believed that a sure measure for our estimates would be furnished us as soon as the collation of an adequate number of individual cases had thrown light upon the actual working capacity of eyes that had been injured. It was therefore determined statistically how much the workmen, whose eyes had been injured, earned prior to the accident, how much immediately after the injury, and how much at a later period. These statistics demonstrated that exactly the same injury and functional disturbance caused a considerable loss in wages to one workman, but not to another. The natural corollary of this fact is that the wage earned after the accident is not alone sufficient for a basis from which to estimate the wage earning power. How can an exact percentage calculation of the loss of earning power be guaranteed from statistics that show that after a certain serious injury of the eye, one workman perhaps continues to work for the same wages for a long time and then has to quit, that a second works on in the same place at a reduced wage, that a third changes his occupation, and that a fourth goes elsewhere and earns more than he did before the accident? Every ophthalmologist who has had anything to do with the national economy, who has himself considered the immense multiplicity of vocations in the business world, knows that factors enter into amount of wages paid, which vary continually and vitiate the calculation.

The social legislation itself has shown us the way out of these many difficulties. It takes no account of the fact that a workman may receive the same wages for a long time in spite of the accident, it aims to insure him in case he can no longer earn so much. A just indemnity indemnifies the person injured for the possible consequences of the accident; therefore it is now generally acknowledged that there is only one way in which we can obtain a satisfactory percentage estimate of the loss of earning power incurred through injuries to the eyes.

4. Estimate of the Loss of Earning Power after Injuries to the Eyes Based on the Loss of One Eye

We must start from the legally recognized principal types of injuries to the eyes, and try to determine in how far the earning power has been reduced. This duty is facilitated by the fact that the sound man is provided with two eyes. What is the earning power when one of them is lost? Monopsia produced by accident must be the starting point for the physician who has to formulate an opinion and must be the basis of his estimate. Granted an agreement concerning the wage loss after such an accident the other possibilities are more easily estimated.

100. The Judicial Opinion in a Case in Which an Eye Has Been Lost, and the Importance of the Artificial Eye

Our duty today is to pass upon a patient who has lost an eye as the result of an injury. Such a case as this forms the basis of all our estimates of the loss of earning power incident upon injuries of the eye, and therefore we must consider what we have to investigate, in order that we may decide most conscientiously how much the earning power of such an individual has been diminished.

The first step is to test the vision of the remaining eye; the vision of the right eye of this patient is to be recorded as perfectly normal. Now we must know to what degree a person with only one eye, that one having normal vision, is at a disadvantage in competition with other men who have two normal eyes, if we wish to estimate the diminution of his earning power.

Investigations have shown that the monocular central vision is, in the first place somewhat less than the binocular, and in the second place, that, after the loss of one eye, the optical pictures have lost something of their clearness. This may be sufficient to render a person with only one eye unfit for certain callings, such as the railroad service. Moreover, a person with only one eye has lost a sixth of his visual field, or perhaps more, if the eye is deeply seated and the bridge of the nose is high, and this may be of importance in many fields of labor, as, for example, on scaffolds, or among the wheels of a machine, but the most grievous trouble of the person who has lost an eye is the loss of binocular vision.

This brings us to the second point, which should never be omitted in an examination of a patient whose eye has been injured; the test of the binocular vision. We shall consider the great importance of the binocular act of vision from a scientific standpoint in our studies of strabismus, but now I ask that each student convince himself practically, by the occlusion of one eye, what the loss of an eye signifies to the stereoscopic vision of our patient. Then only can such cases be rightly appreciated.

We use in practice to test the binocular vision *Hering's* experiment with falling bodies and the beautiful rod test. *Pfalz* has devised a stereoscoptometer which furnishes us at the same time with numerical data concerning the degree of disturbance. Such a patient as this has lost the power of judging proportionate sizes, of estimating distances and the finer perceptions of depth. This one complains that when he is trying to drive a nail he frequently fails to strike it. The loss of binocular vision is of great importance in many trades, as in mining, in factories and in the fine mechanical arts, and must be taken into serious account.

It has been claimed that a person with one eye becomes accustomed to the monopsia, learns to overcome his trouble to a certain extent and acquires a monocular perception of depth, but we must make a distinction in this regard between persons who lost an eye in early childhood and those who suddenly suffered this loss in later life. The former, when they have intelligence and inclination, can, in fact, partially compensate for the loss of the binocular perception of depth; they learn to judge distances and the relations of depth and size with approximate correctness, through a refined and expert muscle and innervation sense of the one eye. It is much more difficult for older persons who have lost an eye to attain this power. Some ophthalmologists have observed a partial return of the power of estimating depth in the course of the first year after the injury, but this has not been observed by others. The instantaneous estimate of depth is, at all events, permanently lost, though habituation may go so far, through practice and the development of the muscle sense, that the absence of binocular vision may not be a disturbing factor in many trades. Nevertheless, persons with only one eye are permanently excluded from callings that make high demands on stereoscopic vision. I do not advocate a diminution of the indemnity when a certain training and habituation has been attained, because the reduction could be for only an insignificant amount and would serve simply to exasperate the victim.

Persons who have lost an eye complain that they are often obliged to change their occupations, to exchange a position that brings them a good wage for one that pays less. Not infrequently they are discharged by their employers as unfit for the work they formerly performed. Others are unwilling to expose the remaining eye to the danger of injury. In many cases the change of occupation must be considered justified, and this must be taken into account in the indemnity. Furthermore, we must not forget that a person with only one eye has less ability to compete in the labor market. We often hear complaints that such unfortunates can find no work, that employers do not, frequently cannot, employ them, because they think such employees will be exposed to danger. Other employers hire them for only a small wage. Finally it should be mentioned that many people with only one eye complain of a lack of endurance while working, that they quickly become tired.

With a knowledge of these facts the first thought is that if this person had perfect earning power with two normal eyes he will have lost one half of his earning power with the loss of an eye and is entitled to an indemnity of 50%. Such an estimate was made repeatedly by ophthalmologists just after the enactment of the accident insurance law, but it was soon perceived that such an award involved great injustice. In order to determine how much earning power the individual who had been deprived of an eye had really lost, statistics were collated showing how much those who remained in the same occupation were still able to earn at a later period. These statistics showed, for example among miners, that half of them continued to receive the same wage as before the injury. To pay them an indemnity of 50% was unjust. Other statistics showed a loss in wages of only from 7 to 15% of the normal. But it must be clear to every intelligent person that the wage paid is not a reliable measure of the actual earning power, because wages depend on many fluctuating factors, such as the condition of the labor market. The purpose of the social legislation is to assure the one eyed workman an income that will secure him against the worst possibilities, hence the decision of the Imperial Insurance Office is as follows:

When required to pass upon a person whose eye has been injured we have first to inquire whether he is a qualified workman or not. By qualified workmen we understand, according to the instructions of the Imperial Insurance Office, such as either need particularly accurate binocular vision from the nature of their occupation, or are exposed in their work to dangers for the avoidance of which unrestricted vision is necessary. The Imperial Insurance Office has fixed the indemnity for the loss of an eye at 25% for the unqualified workman, and at from 30 to 35% for the qualified. Physicians must abide by this decision, which is in harmony with the results obtained by the researches of many ophthalmologists in recent times. With regard to the question whether habituation may justify a reduction of the indemnity I will say again that this award is not excessive. This habituation is very questionable, for it can never replace much that has been lost. Many trades continually require accurate estimates of distances, dimensions of depth and conditions of level, among which may be mentioned carpenters, smiths, machinists, molders, miners, mechanicians and chemists. Above all the conditions of labor may change any day, for the one eyed workman, and many trades are closed against him. The general welfare is the only object of the social legislation.

When we discharge a patient, after the removal of his eye, we furnish him with a suitable prothesis.

Healing usually takes place rapidly and smoothly after an enucleation. At first there is a slight swelling of the conjunctiva which is maintained by hemorrhages in the orbit. We remove the sutures on the fourth, or fifth, day, wash out the conjunctival sac frequently with a solution of boric acid and try to insert the prothesis at the end of a week or ten days.

After an exenteration of a suppurating eyeball there is usually an inflammatory swelling of the lids and a great chemosis of the conjunctiva, which may persist for days, or even weeks. Ordinarily the conjunctival secretion is increased. It also takes some time for the sclera to become folded together into a stump and for the blood, which sometimes accumulates in its cavity, to become organized, so that several weeks usually elapse after an exenteration before the prothesis can be inserted. The disadvantage that the healing is delayed after an exenteration is compensated for, as a rule, by the fact that the prothesis moves better in association with the movements of the good eye, and so the cosmetic effect is improved. The better mobility is due to the fact that the prothesis rests on a stump to which the muscles are still attached. Still, enough can be done for the cosmetic need after an enucleation. Of course it is necessary that the conjunctiva be dissected up close to the margin of the cornea at the time of operation, so as to save as much of it as possible.

A prothesis is primarily worn for cosmetic reasons. It is only right that such patients should have their disfigurement obviated as much as possible, but there are other reasons that not only justify, but compel the wearing of an artificial eye. Workmen with artificial eyes can obtain positions more easily than those who display an exophthalmos. More than that the wearing of the prothesis must be regarded as a direct therapeutic measure. When the conjunctival sac remains empty and exposed to the air a chronic state of irritation becomes developed in a short time, together with more or less secretion. Not only is such a conjunctival catarrh very annoying, but the secretion may infect and endanger the conjunctiva of the other eye, as it can be transferred very readily by the hands, or by handkerchiefs, or during sleep. The prothesis forms an efficient protection of the orbit from the effects of air and dust, and is therefore of therapeutic importance.

If no prothesis is worn after an eye has been enucleated the conjunctiva shrinks considerably. When a child neglects to wear the prothesis the growth of the orbit and the development of the side of the face may be retarded. The artificial eye must be neither too large, nor too small, and must correspond well in color and size of pupil to the other eye. Rights and lefts are furnished. After the prothesis has been chosen a few hints should be given the patient, who will quickly learn to insert it himself. It should be removed during the evening and placed in clean water for the night. Even the best prothesis becomes useless in time, because the tears gradually roughen the surface of the glass. Artificial eyes need to be renewed on the average every year or two. The best ones are the "reform" eyes.

101. Judicial Expression of the Diminution of the Earning Power by Reduction of the Central Vision of One Eye and by Monolateral Aphakia

We began with the simplest case in which a medical opinion is called for: total loss of one eye with preservation of the other in a healthy condition. The Imperial Insurance Office has established in such cases an indemnity of $33\frac{1}{3}\%$ for qualified workmen, of 25% for the unqualified. We have also to furnish estimates when an injured eye is not lost, but its vision has been partially destroyed. Such cases are very common and are exemplified by these two patients.

In our first patient we find that one eye is normal, while in front of the pupil of the other, occupying the central portion of the cornea, is a roundish, old opacity, of aponeurotic whiteness, which was caused by a traumatic ulcer. The opacity is pretty sharply circumscribed in this case, it does not completely cover the pupil, and the eye still has a vision of 0.1 of the normal.

In our second patient the lens was injured by a sharp instrument through a small, peripheral wound in the cornea. He developed a traumatic cataract, which was removed by operation. The vision of the uninjured eye is perfect, that of the aphakic eye is less than 0.1 of the normal without a glass, but more than half the normal with the proper correcting lens.

An accident indemnity is to be awarded to each patient, and the question is how to estimate the diminution of the earning power. I have intentionally presented these two cases together, because, whenever it is necessary to furnish an estimate of the diminution of the earning power in only partial impairment of the vision of one eye, the cases fall practically into two groups, one group comprising those in which the central vision is reduced, while the lens and the mechanism of accommodation is preserved; the other, those of monolateral aphakia caused by accident.

The central vision of one eye may be impaired by opacities in the cornea, or the other refractive media, of traumatic origin, or perhaps by such conditions as traumatic dilatation of the pupil, retinal hemorrhage in the region of the macula, or rupture of the chorioid, after a slight, or a severe contusion. All such impairments of the central vision may reduce the earning power in many different trades, and the question arises: What relation does the reduction of the earning power bear to the impairment of the central vision? Originally it was thought that this relation was directly proportional, and that it could be expressed by mathematical formulæ. This was an error. The earning power of persons who have one good eye and one with only a portion of its central vision does not decrease in practical life proportionately with the vision. There are certain callings which make great demands on the two eyes, in which the function of the good eye is interfered with by the functionating of the injured one, as in the railroad service, or the navy. In most trades the necessary work can be done with less than full, scientific vision. On the other hand, a man may be very far from being blind and yet may be totally incapacitated for work.

These facts have given rise to the concept of the working vision, the upper and lower limits of which are narrower than those of scientific vision. These limits we have to determine, but generally they cannot be calculated mathematically.

We understand as the upper limit of the working vision that portion of the scientific vision which is sufficient for the undisturbed and perfect performance of the work. The lower limit is that degree of scientific vision at which the work cannot be performed. It is evident that these limits must vary a great deal in the different trades. A laborer who does only coarse work, like tilling the soil, can do his work with a vision that is not sufficient for a fine mechanician. All manner of attempts have been made to ascertain these limits of working vision for the different trades, but the influential factors are so manifold, and often so confusing, that no mathematical formulæ can be established either for the trade or for the individual.

In a manner corresponding to the division of laborers by the Imperial Insurance Office into qualified and unqualified workmen, we divide the trades into two groups, for practical reasons; those which make higher, and those that make lower optical demands on the workmen. We take as the upper limit of the working vision $\frac{3}{4}$ of the normal in trades that make great demands on the eyes, $\frac{1}{2}$ the normal in those that make less demand. The lower limit is $\frac{1}{6}$ of the normal in the former, $\frac{1}{10}$ in the latter.

An example will show that no simple proportion exists between the lowering of the working vision and the decrease of earning power. Suppose that a man who has lost an eye, like one of our former patients, is employed in labor that makes high optical demands; a slight injury to the remaining eye will suddenly incapacitate him for doing the finer work. It happens very often that the earning power does not sink uniformly with the reduction of the working vision, but gives way suddenly.

In the overwhelming majority of cases, impaired vision in one eye with normal vision in the other diminishes the earning power only when the stereoscopic vision is destroyed. Investigations have shown that, when the refraction of the two eyes is the same and their position normal, binocular vision and perception of depth are still present when the vision is reduced to $\frac{1}{10}$ of the normal, frequently when it is still less. Our first patient has good perception of depth, and it might be thought at first that a patient with one normal eye and binocular perception of depth has not suffered any material loss of his earning power in spite of the impaired vision of the other eye, but it would be wrong to refuse him an indemnity, because, while the preservation of the binocular vision is the first, it is not the only factor that enters into our computation. In fact many other troubles have to be taken into account in a monolateral impairment of the central vision. The irregular refraction produced by the ill defined margins of corneal maculæ and of opacities of the other media may cause a considerable degree of dazzling. Cicatrices in the cornea produce irregular astigmatism, with its troubles. The pupils may be altered in form and size by traumatic mydriasis, leucoma adhærens, or coloboma. We must also bear in mind that the person injured must work under conditions that are quite different from those of our ordinary visual tests. Hence an indemnity must be granted.

An indemnity of 10% is awarded for a reduction of the vision to between $\frac{1}{3}$ and $\frac{1}{6}$ in qualified workmen, to between $\frac{1}{4}$ and $\frac{1}{10}$ in unqualified. Whether the indemnity may be discontinued after one or more years must be decided in each case. This depends on whether a real improvement has taken place and on whether the person has demonstrably lost nothing of his earning power. If the vision of the injured eye is less than $\frac{1}{10}$ the indemnity may be raised to 15 or 20%, because the condition now approximates that of blindness, and only the binocular field of vision is of use. When fingers can be seen only at a short distance we look upon the eye as practically blind.

When we have to deal with monolateral aphakia, as in our second patient, we have to base our judgment in part on altogether different grounds. This man likewise has an impairment of the central vision of one eye, which certainly seems to be obviated by the cataract glass. He tells us, however, that he cannot wear the glass; with the best will in the world he cannot work with it on, for reasons that were given in detail in the study of monolateral aphakia after cataract operations.

The loss of the fine, binocular perception of depth is of quite serious consequence to the person who has lost the lens of one eye. Even when the central vision is raised to normal by the correcting lens the binocular perception of depth is lost, or is extremely faulty. The great difference in the refraction of the two eyes in monolateral aphakia abrogates the certainty of the binocular estimate of depth. Much more so is this the case when the eye is strabismic, as often happens. The difference is remarkable when this case is compared with the other, in which the vision is impaired, but the eye still contains the lens. It may happen that a person with monolateral aphakia may gradually become habituated to the condition and acquire a monocular estimate of depth, but this is to be expected only in young and intelligent persons. A workman with monolateral aphakia is useless in trades that call for accurate and binocular perception of depth.

The loss of the power of accommodation must also be taken into account in calculating the indemnity in these cases. Persons whose central vision is moderately reduced, as by opacities in the media, can often continue to use their eyes for near work, while those with monocular aphakia are again at a disadvantage, they must constantly change their glasses as they wish to see objects at a distance, or near at hand. Finally, it is not at all uncommon for these people to complain of an annoying dazzling due to the indistinctness of the optic images, especially when the form of the pupil has been changed. It is for this reason that they often cover the eye with an opaque glass, or a patch.

For these reasons it is just that a person who has been rendered aphakic in one eye by an injury should receive an accident indemnity. We maintain that the award should be more than for a simple impairment of vision in one eye, because the condition of the patient is often worse, and generally place it at 15 to 20%. This indemnity is to be awarded even when good vision is obtained with a cataract glass, either primarily, or after an operation for secondary cataract.

102. Judicial Opinion when the Vision of Both Eyes has been Impaired by an Injury and when Other Complications are Present

Thus far we have been studying some of the basal types of injuries of the eye on which medical opinions have to be rendered. It is rather more difficult to estimate the indemnity when the vision of *both* eyes has been impaired, no matter whether the two eyes were injured simultaneously, whether an interval elapsed between the two injuries, or the uninjured eye exhibits an imperfection of vision that is independent of the accident. Our judgment may be influenced also by other complications.

Case 1.—This patient lost an eye some years ago from an ulcus serpens, and has now been attacked by an ulcus serpens in his other eye, an occurrence that is not altogether rare. The ulcer has been brought to a standstill and the eye still possesses 0.2 of normal vision. The patient has been receiving 25% indemnity for the loss of one eye and the question for us to decide is how high to estimate now the loss of his earning power.

Case 2.—Both eyes of this patient were injured in a premature explosion in a quarry. Many grains of powder are to be seen encapsuled in the skin, conjunctiva and sclera. The essential cause of the reduction of vision is the presence of dense opacities of the corneæ. The vision of the right eye is 0.1, that of the left 0.3 of the normal. What accident indemnity should this patient receive?

Case 3.—The vision of this patient's left eye has been very bad ever since he can remember; the eye diverges and exhibits minute maculæ, mementos of inflammations during childhood. In an accident his right cornea was perforated and a traumatic cataract produced. The wound healed aseptically and the traumatic cataract has been removed by operation. With a cataract glass the vision of the injured eye is now $\frac{1}{4}$ of the normal. How much is the earning power reduced in this case?

Every possible combination may be met with in these cases. On the whole it is rare that both eyes are injured simultaneously in an accident; such an occurrence is met with only in explosions (Case 2), shot wounds and accidents with chemicals. It is also comparatively rare that one eye is injured in an accident and, at a later period, the other eye suffers in the same manner. In the majority of cases in which the vision of both eyes is found to be imperfect after an accident, that of one was impaired previously to and independently of the accident. All possible factors come into consideration in the impairment of the central vision of both eyes. We must take cognizance of the refraction, of any strabismus that may be present, of anisometropia, etc. We must know under what conditions of light the injured person has to work, and we must take into account the troubles caused by the dazzling and obscuration produced by corneal opacities, as well as those due to irritative conditions. Experience has shown that it is difficult for physicians to express themselves within the given limits, when passing upon cases with impaired vision of both eyes.

The task is easiest when the second eye has been injured subsequently to the *loss, or blinding of the first.* In such a case the indemnity must be between that for monopsia and 100%. It is more difficult when both eyes have been equally, or unequally, injured by an accident (Case 2), and the diminution of the earning power demands quite a deliberation when one eye was not good before the accident, as in our third case. A workman has very often had one or both eyes impaired before an accident. Think of the cases of corneal opacity that remain from childhood. People with such opacities frequently work just as well as others whose eyes are perfectly healthy, and I advise that, in such cases, the decision as to the working capacity be not based on a presumed impairment of the vision, but rather to consider an eye to have been perfectly normal, when there is doubt as to whether it was normal or not before the accident. To determine in how far the impairment of the eyes before the accident influences the estimate of the indemnity we can start from the fact that the indemnity in a case of total incapacity is $66\frac{2}{3}$ % (full compensation) of the yearly wage for as long a period as the disability lasts. If the incapacity to work is partial, the indemnity should be such a part of the full compensation as corresponds to the loss of earning power. The condition of the injured person before the accident must be compared with his condition afterward, and we have to ascertain what part of his individual earning power has been lost through the accident. If his earning power, as compared with that of a sound workman, has suffered a certain percentage of impairment, say by monopsia, the annuity he is receiving cannot be lessened. A deficient workman commands a smaller yearly wage, the full compensation is computed on the wage received at the time of the accident, and the indemnity to be awarded corresponds to the diminished wage incident upon the lessened earning power. If a person with monopsia loses his only remaining eye he loses 100% of the earning power he retained after the loss of the first, although this is less than it was originally. Various tables have been computed to serve as guides in fixing the indemnity in any particular case. I recommend the empirically constructed tariff of *Maschke* for unqualified workmen. It serves for trades in which high optical demands are not made and is based altogether on the impairment of the central vision. It is very useful in practice. and we will determine by it the approximate indemnity in each of our cases.

Tariff of Indemnities according to Maschke.

S = ‖	1—2/3	1/2	1/3	1/4	1/5	1/7	1/10	1/15	1/20	0
1—2/3	0	0	5	10	10'	15	15'	20	20'	25
1/2	0	5	10	10'	15	20	25	25	30	35
1/3	5	10	25	25'	30	30'	35	40	45	55
1/4	10	10'	25'	40	40'	45	50	55	60	65
1/5	10'	15	30	40'	55	60	65	70	75	80
1/7	15	20	30'	45	60	70	75	80	85	90
1/10	15'	25	35	50	65	75	85	90	95	105
1/15	20	25	40	55	70	80	90	95	100	115
1/20	20'	30	45	60	75	85	95	100	110	125
0	25	35	55	65	80	90	105	115	125	125

[The apostrophes following the figures indicate that this number is a mean value between the adjacent numbers, which may be increased a little in accordance with other conditions.]

Case 1.—Right eye, amaurosis.
Left eye, vision $\frac{1}{5}$. Indemnity approximates 80%.
Case 2.—Right eye, vision $\frac{1}{10}$.
Left eye, vision $\frac{1}{3}$. Indemnity 35%.
Case 3.—Right eye, vision with cataract glass $\frac{1}{4}$.
Left eye, high degree of amblyopia, vision 0. Indemnity 65 to 75%.

Many associated symptoms need to be considered if we are to form a correct opinion as to the effect of an injury of an eye upon the earning power. Very often the injured eye remains irritable for a long time. This is particularly annoying to workmen who are obliged to labor in the open air, in heat and dust, in the sunshine, by open fires and in the wind. Such eyes are very sensitive and apt to suffer from photophobia, increased lacrymal secretion, pain and other troubles. The causes of this irritability are manifold. A conjunctival hyperæmia persists for a long time after burns; cicatricial contractions, symblepharon, trichiasis and entropion may increase the hyperæmia to a true catarrh. Not infrequently inflammations of the lacrymal sac coexist. Sometimes an artificial eye cannot be worn at first, in spite of the patient's desire to wear it, and the catarrh of the orbit infects the other eye. Other patients, who have adherent cicatrices, or wounds in the region of the ciliary body, complain at times of pain.

The dazzling produced by opacities in the refractive media, or by dilatations of the pupil, frequently deserves an accident indemnity more than the actual impairment of the vision. Benefit may often be obtained by tattooing translucent maculæ of the cornea, so as to obviate the dazzling and to improve the vision by making the edges of the opacity more sharply defined.

All irritative conditions justify an increase of the indemnity 5 or 10%, even though but temporarily, for such persons must frequently abbreviate their hours of labor. The examination needs to be carefully made in such cases, for many patients exaggerate their troubles. Disturbances of the color sense and of the light sense are of less importance in injuries of the eyes. A reduction of the color sense alone can scarcely come up for consideration, and when the light sense is impaired it is usually because of serious changes within the eye of the retina and chorioid, in which case the disturbance of the central and peripheral vision is the essential factor in the estimate of the indemnity. In siderosis bulbi alone can a permanent hemeralopia remain after extraction of a spicule of iron.

Certain disturbances of the visual field and pareses of the ocular muscles have also to be taken into account. It has already been stated that the visual field is contracted about 30 degrees on the side of the injury whenever an eye is lost or rendered blind. This deficiency is negligible in many occupations, but in others it is of serious consequence and occasionally must be appraised among miners, machinists and railroad employees. Isolated contractions of the visual field due to traumatism do not come practically into account. The concentric contraction of the field in traumatic neurosis does not interfere with the earning power, even when an indemnity is awarded because of the neurosis itself. It causes no disturbance of orientation, because it represents only a deficiency in consciousness. Most contractions of the visual field following injuries are associated with an impairment of the vision and therefore call for no separate consideration, as regards the indemnity.

Central scotomata, met with after injuries, whether due to disease of the macula or of the optic nerve, are of practical importance, as are also the bilateral homonymous and heteronymous hemianopsias met with after serious fractures of the skull. These cases require a careful examination and the services of a specialist.

Pareses of the ocular muscles frequently result from injuries and may reduce the earning power. When the levator palpebræ superioris is paretic and the upper lid hangs down so far as to exclude the eye from the act of vision an indemnity is called for, which, under certain circumstances, may be as great as for the loss of an eye. When the pupil is not covered the earning power is not impaired. The consequences of mydriasis due to paresis of the sphincter are dazzling, indistinctness of the retinal images and, sometimes, impaired vision. When the degree is slight I do not recognize any diminution of the earning power, yet in certain conditions, in certain kinds of work and when bilateral, it may need to be taken into account. In monolateral paresis of the accommodation binocular vision may be interfered with, especially when the refraction of the two eyes is different and the occupation is one that makes high demands on the eyes. In such cases an indemnity may be indicated.

The troubles due to traumatic pareses of the external muscles of the eye are far more important. Such pareses may impair the binocular act of vision, and cause diplopia and strabismus, which often reduce the earning power considerably. The paresis of the individual muscles is to be judged differently in this respect. Diplopia in the lower part of the field is very annoying in near work, while in other callings diplopia on looking upward causes the more trouble. The affected eye must often be excluded from the act of vision, and change of occupation may be necessary. Every such case must be passed on individually in regard to the estimate of the indemnity.

103. Relations of Injuries of the Eye to Some Infectious Diseases

Questions concerning the connection between an injury and all possible diseases of the eye continually arise, for patients who have had an eye injured are apt to refer to the injury all of the ailments that may later afflict that organ.

A case in point is trachoma. It frequently happens in Germany that claims for accident insurance are made by persons suffering from trachoma, which is known to be an essentially chronic, infectious disease of the conjunctiva, specific for human beings, with an active agent in the conjunctival secretion, and the question may be formulated thus: In how far is this statement, that trachoma may be caused by an accident, justified by the nature and the clinical course of the disease? We have here a patient who states that some time ago dust flew into his eyes while he was at work in the field, that prior to that accident his eyes were healthy, and that ever since then his right eye has been sore. Examination reveals, however, signs of a trachoma that has lasted a long time not only in his right eye, but in his left as well. He lives with other field hands, some of whom also have trachoma. We know that foreign bodies carry no trachoma germs on them. For these reasons we deny the claim to an accident indemnity.

Another possibility that has been suggested is that trachoma may occasionally be the indirect consequence of an accident. I am very sceptical in regard to this point. I could recognize a causal connection only in a case in which it had been determined by a physician immediately after the accident that the injured eye was not trachomatous, and the subsequent development of a monolateral granulosis rendered the indirect transmission of the disease probable.

The conditions in our second case are quite different. His trachoma had improved sufficiently under treatment to enable him to return to work; he has now suffered an injury to the eye and the disease has been rekindled. There is no doubt that a trachoma which has been apparently cured may be made worse again by an injury, so when this patient files a claim for accident indemnity we will support it. The only precondition is the positive proof of the accident.

Sometimes it happens that recovery from trachoma is rendered difficult and is delayed by an accident. Dust or mortar in an eye may aggravate the trachomatous conjunctiva and retard the improvement. The danger of infection is also greater when such eyes suffer from perforating wounds, and we often find it difficult to perform plastic operations on them. The cornea is particularly endangered in trachoma by pannus, which may be aggravated, or rekindled by an injury. In such cases the accident must be proven positively, and it must not be forgotten that exacerbations and recurrences of trachomatous pannus are of daily occurrence, even in the absence of injuries.

It is quite difficult for a physician, who is called upon to formulate an opinion, to decide whether a corneal ulcer in a patient who has trachoma is to be ascribed to an injury, for in many cases such an ulcer is due to the trachomatous infection of the cornea and is the cause of the gravity of the disease. At the same time it is of course quite possible for eyes with trachomatous corneal opacities, the vision of which is not good, to suffer from superficial injuries. The differentiation is particularly difficult when the claim for indemnity is made by the patient a long time after the disease has run its course. In such cases it is important to compare the two eyes to see whether one or several leucomata are present, for in this way a diagnosis of greater or less probability can often be made. In only about one third of all the trachoma patients who file claims for indemnity can a causal connection between accident and trachoma be considered probable.

We have rather more data to aid us in the differentiation of trachomatous from traumatic ulcers, when ulceration is present in a trachomatous cornea. We know that the typical ulcus serpens, which cannot be produced except through an injury, is met with very rarely in the trachomatous cornea, in spite of the fact that we very often meet with dacryocystitis in trachoma. Evidently the pneumococci do not obtain a foothold easily in the vascularized cornea. If pneumococci are not found in such a fresh ulcer of the cornea, the probability that the ulcer is traumatic is not increased, although such an etiology is not absolutely excluded, for diplobacilli and other agents can produce ulcers of the cornea after superficial injuries, but negative bacteriological findings increase the improbability that the ascribed injury was the cause. Here we have a patient that illustrates the point. This man has a trachomatous pannus in both eyes and a horizontal oval ulcer on one cornea, such as is met with in trachoma, which he ascribes to an injury; but the time when the accident happened does not harmonize well with the development of the ulcer, and the bacteriological condition of the latter is negative. We have also some clinical signs, in addition to the bacteriological examination, by means of which traumatic and trachomatous ulcers of the cornea may be distinguished. It has been observed that the development of vessels from the limbus is much less in traumatic ulcers than it is in trachomatous, which are usually accompanied by a subepithelial vascularization. All of these things have to be taken into account when we are obliged to consider the causal relations between trachoma and injuries of the eye.

We will turn now to other infectious diseases and to their connection with injuries of the eye. Tuberculosis demands attention first, as it does everywhere. We know from our studies of diseases of the conjunctiva that insignificant lesions of that membrane evidently permit ectogenous infection with tubercle bacilli in genuine conjunctival tuberculosis. Cases have also been reported in which it was demonstrated that tuberculosis of the conjunctiva developed in immediate connection with injuries and operations for strabismus. The entrance of tubercle bacilli may be rendered possible by foreign bodies, especially into the tarsal conjunctiva of the upper lid. Primary tuberculosis of the cornea after a superficial injury is more rare.

The relation of the frequent intraocular tuberculosis to injuries has to be taken far more into account. The bacilli may be carried directly into the eye by a perforating wound and so bring about a clinical picture analogous to those that we frequently produce for diagnostic purposes by inoculations in the eyes of animals; but tubercle bacilli may also be carried by the blood from other foci of the disease in the body to the eye, and may settle there when the eye has been injured by a contusion. We cannot recognize a causal connection with an alleged former slight contusion in every case of tuberculous iritis without additional evidence. The conditions as to time and the entire course of the disease must be such as to render an etiological relation probable. I am particularly sceptical concerning the frequently alleged connection between traumatism and tuberculous interstitial keratitis. We have far better surgical analogies for the etiology of tuberculosis of the margin of the orbit, where a traumatism may very easily create a locus minoris resistentiæ, and prepare the way for an endogenous tuberculosis, and in suitable cases it is evident that an ectogenous tuberculosis of the margin of the orbit may be produced.

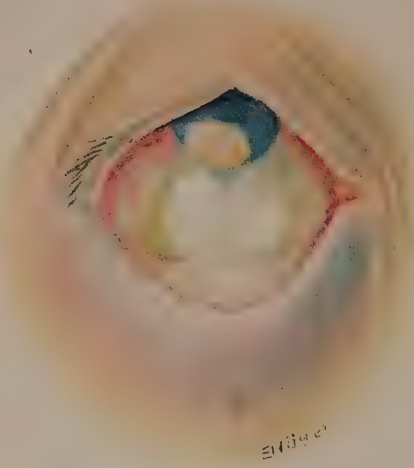
After tuberculosis comes *syphilis*, a disease which is of considerable importance, as we have learned while studying diseases of the cornea and of the iris. It has been ascertained in numerous cases of primary affection of the eye, which is usually situated on the lids, or brows, less commonly on the conjunctiva, that the germs were inoculated through injuries. In how far an injury of the eye has any influence upon the appearance and development of syphilitic diseases of that organ in cases of general syphilis, whether hereditary or acquired, has not yet been sufficiently investigated. The only question of interest in this respect is whether a traumatism to an eye may excite a typical keratitis parenchymatosa. It may always be claimed that a traumatism to a person suffering from general syphilis may excite the disease endogenously at the place injured, but keratitis parenchymatosa, as an accompanying symptom of hereditary syphilis, is usually bilateral and attacks the second eye some time after its appearance in the first, so the question really is whether we have any basis for the idea that a traumatism, either slight or severe, to one eye can excite a syphilitic disease such that the second eye will become affected weeks or months after the infection of the first. Can a slight injury of one eye cause an endogenous disease of the other weeks or months later? The suggestion has been made that the accident to the eye may excite vasomotor irritations which may induce the circulating microorganisms to settle, but the most recent experimental researches concerning the effect and transmission of irritation as regards the eye have shown that the irritation of one eye is unable to cause the least secretory change in the other. When we examine critically the clinical material that has been used hitherto as a basis for the theory of a causal connection between traumatism and bilateral parenchymatous keratitis, we find that no such causal connection can be demonstrated. I think that in these cases the accident indemnity has been carried too far, and I maintain that no connection between traumatism and bilateral keratitis parenchymatosa can be recognized. We ophthalmologists have to depend very much on the cooperative work of the general practitioner in regard to these matters. In cases that may later be contested the eye should be examined immediately after the alleged injury by the physician, and it should be ascertained whether the cornea was affected by the traumatism, and whether both eyes were perfectly healthy at the time of the accident. When this is done we will see how many of the cases in which a traumatism is held responsible for the later development of a bilateral syphilitic parenchymatous keratitis can be proven.

I recommend like precautions in the formation of a judgment in a case of acquired syphilis, when the question arises whether a traumatism has excited the syphilitic disease in the eye. Insignificant injuries of the conjunctiva by particles of dust have been held responsible by patients for a syphilitic atrophy of the optic nerve.

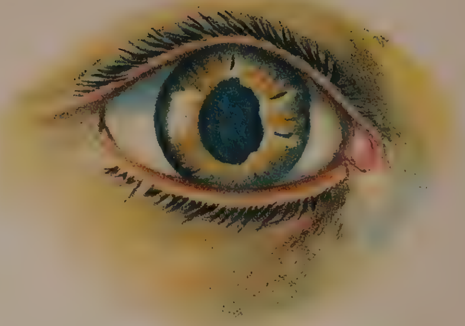
Another serious disease that may follow an injury to the eye is *tetanus.* It is well that physicians should bear this disease in mind whenever they have to deal with fresh, severe injuries of the eye, or of its environment, although cases are rare. Tetanus is seldom to be feared after an injury to the eyeball, but is more apt to occur after injuries to the lids and orbit, especially when the object with which the injury was produced has been in contact with the ground. Dirty wood and earth contain the spores of tetanus bacilli. The disease takes the cephalic form in cases of injury to the eye, and the period of incubation fluctuates between one and ten days. The diagnosis is made easily when dysphagia, trismus and facial paresis are present. Trismus must not be overlooked when there is a great inflammatory swelling of the face. The prognosis in these cases is very bad, partly because of the peculiar nature of the locality where infection has taken place, partly because it has been impossible to administer antitoxin soon enough in most cases. When a severe and dirty case of injury of the orbit comes under treatment our first precaution should be to inject the serum, afterward to cleanse the wound thoroughly, to lay bare the orbit, and to remove the foreign body. Persons who have been thus injured should not be sent a long distance to a hospital until they have received an injection of serum, for the antitoxin protects more effectively than it cures.

Another disease that may follow an injury to the eye is *anthrax.* This usually starts in the lids, rarely in the mucous membrane, and appears either as a carbuncle, or in the characteristic form of œdema. The anthrax carbuncle begins as a little red nodule, which soon develops into a pustule having greatly swollen and infiltrated margins. A black eschar soon forms on the site of the pustule, the carbuncle enlarges rapidly and the gangrene of the skin extends. By this time the lids are so swollen that it is very difficult for us to obtain a view of the eyeball. The neighboring lymphatic glands become swollen. In a mild case the necrotic portions, which often extend very deeply into the tissues, slough away, and the subsequent cicatrization causes ectropion of the lids, as a rule, which will need to be relieved later by an operation. In a more serious case the carbuncle continues to enlarge and cellulitis appears in its neighborhood, associated with grave general symptoms, fever and great prostration. When the agent is very virulent a general infection takes place and the patient dies of anthrax sepsis. The simple or grave furuncle, or carbuncle must be differentiated in such a case. The vocation of the patient frequently furnishes an indication, for the people who, above all others, are subject to anthrax are those who deal with animals, or their products. The diagnosis is made positive by the demonstration of the anthrax bacilli, which may easily be found at the margin of the tissue necrosis.

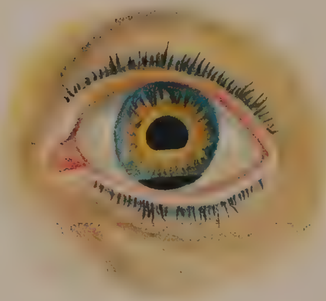
The second form of this disease in the eye is the œdema of anthrax. An extensive œdema of doughy consistence develops in connection with a little pustule. Large blebs, with serosanguineous contents, may appear on the skin. The prognosis is bad from the start. The œdema spreads rapidly over the cheek and face to the neck and the body, and a general infection quickly supervenes. This œdema may be mistaken for a progressive cellulitis. The mortality is between 5 and 20%. An injection of anthrax serum is to be recommended as soon as such a case comes under observation, according to recent experience. The local treatment of an anthrax carbuncle consists of the application of hot cataplasms, that



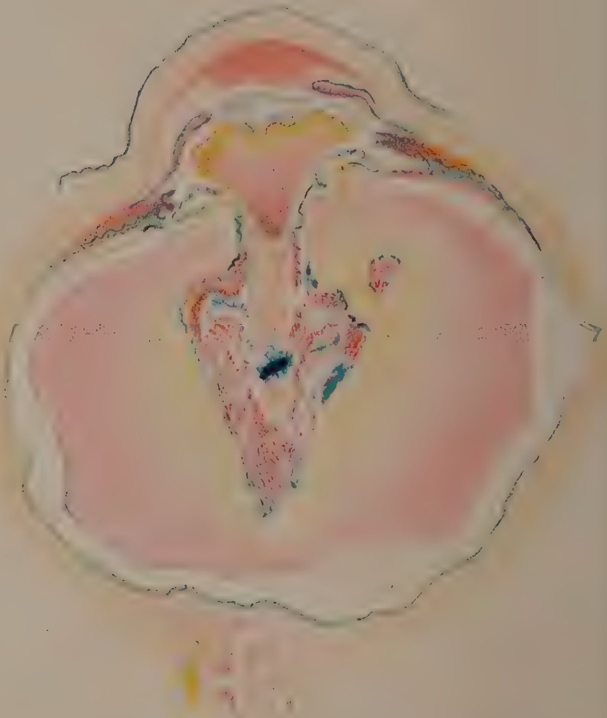
a Recent burn from red-hot iron.



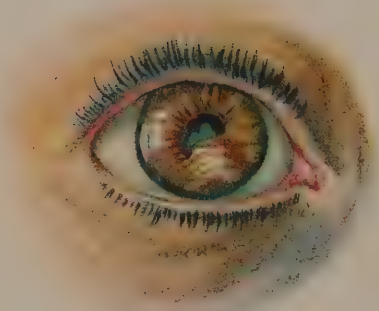
b Traumatic Mydriasis with radiating slits in the surface of the iris.



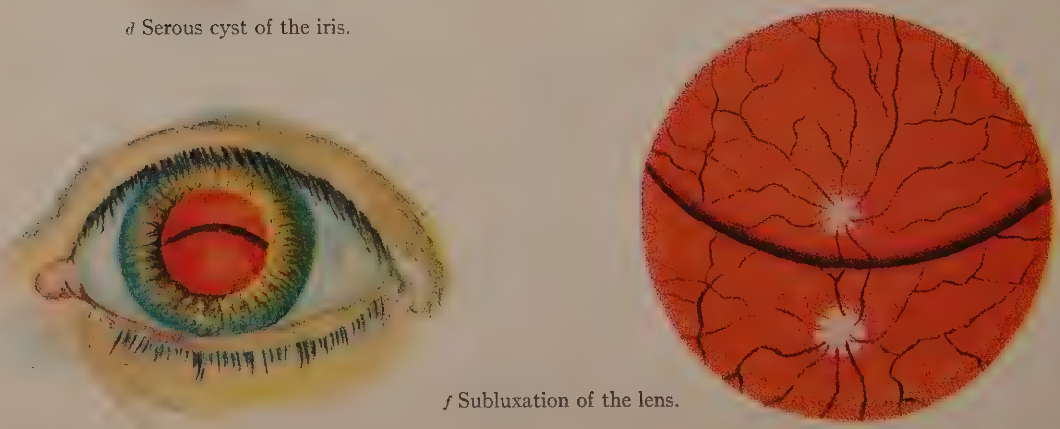
c Iridic dialysis.



e Siderosis bulbi, bits of iron in the vitreous humor.



d Serous cyst of the iris.



f Subluxation of the lens.

of the œdema consists of injections of a 2% solution of carbolic acid. Such cases are of surgical rather than of ophthalmological interest.

Glanders and *rabies* may be mentioned as other grave diseases that have been observed after injuries to the eye. Glanders is met with in persons who have come in contact with animals suffering from this disease; such an infection results in very serious infiltrations, ulcerations and gangrene. Hydrophobia may be communicated by the bite of a rabid dog in the eye. In such a case our duties are confined to the cleansing of the wound and the immediate transference of the patient to an institute for the treatment of hydrophobia.

104. Connection between Injuries and Tumors of the Eye

Many causal relations have been found to exist between traumatism and malignant tumors in surgery and general pathology, so it would seem as though the possibility of such causal relations between injuries and tumors might be assumed in ophthalmology, and very often the patients subsequently ascribe their tumors to traumatism.

We do not yet know the cause and the real nature of the development of tumors, but we certainly do know that an injury is not the only, and not the direct, cause of tumor formation. We can only question in certain cases whether the injury took part in exciting the formation of a tumor, or whether it favored the development of such a growth.

To begin with we must classify the different kinds of tumors of the eye into groups. Intraocular sarcomata first have to be taken into account. Quite a number of observations are recorded in literature to prove the relation between this form of sarcoma and injuries, but a critical sifting of these observations has shown that the conditions are much more unfavorable for a traumatic origin in an intraocular sarcoma than in similar tumors of other superficial organs. The potential germ of the development of sarcoma must have been present in the eye before the injury in all such cases, and therefore the only question that can be discussed is whether the injury took part in, or favored, the development of the tumor. Such a question can never be answered with certainty. I would consider a causal connection between a traumatism and this form of tumor to be probable only when the injury was severe, when acceptable relations between the place of injury and the site of the tumor can be determined, and when the time that elapsed between the traumatism and the appearance of the symptoms of tumor supports the idea that they were connected. The Imperial Insurance Office demands in contested cases the proof not only of an accident, but also that the accident actually affected the place where the tumor developed, and also makes it conditional that a number of troubles and symptoms persisted during the period between the accident and the development of the tumor. When this test is applied we can trace a causal connection back to the many asserted injuries in only a very few cases of intraocular sarcoma.

Traumatism plays no part in the etiology of malignant intraocular glioma.

We are more apt to think of such a causal connection in epibulbar sarcoma and epithelioma. The epibulbar sarcoma develops by predilection in the region of the palpebral fissure, from a nævus of the conjunctiva near the limbus. We know that the region of the palpebral fissure is subject to many little injuries, but here again we must be careful in forming an opinion. The conditions are quite similar in epithelioma, which likewise starts at the limbus. Cases have frequently been described in which a preceding traumatism could be proved. An etiological connection with an injury has also been recognized repeatedly in cases of sarcoma and carcinoma of the lids, and of the lacrymal gland, as well as of the soft tissues and bones of the orbit.

Although we maintain a certain reserve in the formation of an opinion concerning the connection between malignant tumors and traumatism, we must admit that a causal connection between benign tumors and injuries is often present. We see cysts develop after operations, particularly those for strabismus, in which displaced cilia frequently play a part. Still more often granulations and pedicled papillomata originate from injuries and wounds produced by operations. Angiomas of the conjunctiva have also been observed to follow severe injuries of the cephalic vessels.

Cysts of the cornea have been observed after injuries. Chief among these are the cystoid cicatrices that are met with after operations and accidents, which have been fully described among the complications that follow the operation for cataract. Strictly speaking these ectatic cicatrices should not be said to be of the cornea, because they communicate with the interior of the eye, but the important part taken by the traumatism is evident.

A perforating wound at the corneoscleral margin may be followed by the development of a large cyst, lined with epithelium, which is connected with the interior of the eye. The cornea, prolapsed iris, conjunctiva and sclera may be involved, and we may divide these cysts into sclerocorneal and scleral. In some cases the endothelium of the cornea has wandered into and lined the cyst. In others the epithelium of the cornea was implanted by the injury, or has proliferated inward from the wound.

Many benign tumors, which may be ascribed to injuries, are also met with in the tissues about the eyeball. An injury to the lacrymal gland, or to its environment, may cause a cicatricial occlusion of an efferent duct and give rise to a cystic dilatation known as dacryops. A contusion of the margin of the orbit, or a cicatricial contraction of the conjunctiva, may result in such a retention cyst of the lacrymal gland, but further than this the origin of dacryops is still unexplained, for when we try mechanically to occlude one of the ducts, as has been done experimentally many times, the gland atrophies, but no cyst is formed. True tumors of the lacrymal gland following injury are very rare.

Polyps may originate in the lacrymal sac from injuries, such as are produced by the passage of probes.

Secondary osteomata often appear on the margins of the orbits, which are exposed to all possible kinds of injuries. Cysts of the accessory sinuses, that encroach upon the orbit, have been described as following traumatisms, and it may be added, for the sake of completeness, that cavernous angiomata of the orbit and phlebectasiæ have also been associated with injuries.

There still remains one intraocular form of tumor in the origin of which traumatism plays an important part: the cyst of the iris. We distinguish two varieties, the *serous cyst of the iris* and the *pearl cyst*. The former is the more common; according to *Wintersteiner* it is met with in comparison with the latter in the proportion of 125 : 30.

I am able to show you a case of serous cyst of the iris (see Plate IX). This eye was injured by a perforating wound two years ago. The striking symptom now is a roundish, partly flattened tumor, with an evidently delicate, transparent wall and clear contents, in the lower portion of the anterior chamber. This tumor plainly arises from the iris and traces of iris tissue can still be seen in its wall at the place where it joins this membrane, particularly with *Zeiss'* loupe. The patient comes to us now because the eye gives him pain, although it recovered with good vision from the injury. The eyeball is irritated and its tension is distinctly increased. When such a cyst of the iris is left to itself its growth is generally constant, although occasionally it may remain at a standstill for a considerable period. The consequences cannot be avoided. As soon as it reaches the posterior surface of the cornea the latter becomes opaque at the place of contact; the cyst grows larger, extends over the pupil, may press the lens backward, and finally secondary glaucoma develops.

It is only in exceptional cases that the wall of the cyst may rupture and the cyst itself undergo involution. We are therefore compelled to operate on serous cysts of the iris. If the eye still possesses vision we must try to remove the cyst as soon as possible by one of the methods at our disposal. We may simply puncture the cyst, an operation that may need to be repeated many times, or we may incise the cyst wall in a manner similar to that employed in performing iridotomy, or we may make a partial, or a total excision of the cyst wall. The attempt should be made to totally extirpate the cyst if it is small, but such a procedure is impossible after it has reached a large size. Even partial excisions are attended in such cases by considerable danger. In this case we will try first the opening of the cyst, an operation which may have to be repeated.

Several theories have been advanced concerning the pathogenesis of serous cysts of the iris. According to one theory epithelium is dislodged from the cornea or conjunctiva, at the time of the occurrence of a perforating wound, and carried into the iris, where it proliferates and forms a cyst (*Rabl* and *Rothmund*). *Stoelting* thinks that the epithelium of the corneal wound proliferates into the anterior chamber and implants itself in the iris. According to *de Wecker* many such cysts originate through the formation of a sac by folds of the iris and distention of the sac with aqueous. According to others such a sacculation of the iris could take place when its tissue is split, either directly by a laceration, or by hemorrhages; or little hollow spaces may be formed in the iris, through the inflammatory action of foreign bodies, become lined with epithelium and enlarge, after the nature of exudation cysts. Numerous experimental researches have been instituted in order to produce such cysts by the implantation of various kinds of tissue. Serous cysts of the iris have no single cause.

Pearl cysts, which are met with more rarely, are firm tumors in either the tissue, or the surface of the iris. The surface of these tumors has the lustre of mother of pearl. They likewise follow an injury, usually when a cilium has been carried into the anterior chamber, and months or years may elapse after the injury before the tumors begin to grow. They threaten the eye in the same way as the serous cysts. Their pathogenesis has been cleared up by numerous researches. Epithelial cells coming from the epidermis, from the root sheaths and cells of the hair bulbs of the cilia, or from the epithelium of the cornea, are torn away at the time of the accident and transplanted into the iris, where they proliferate.

The only possible treatment is operative, for spontaneous resorption of the implanted cells is rarely brought about by smothering with granulation tissue. A large flap section is necessary for their total extirpation.

VII

Diseases of the Vitreous

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105. The Most Important Opacities of the Vitreous

Diseases of the vitreous can be diagnosed only with the aid of the ophthalmoscope. It is but occasionally that we can perceive morbid changes in its most anterior portion by means of focal illumination. We have met with diseases of the vitreous many times in the course of our clinical studies; I need only recall to mind the intraocular hemorrhages after injuries, and the opacities found in cases of iritis and iridocyclitis. The vitreous plays a rather passive part in diseases of the eye, it is simply a receptacle for pathological products from the surrounding ocular membranes, and yet some typical pictures are presented. I would also like to excite interest in the vitreous, because far too little investigation has been made as yet into the importance of the pathological composition of the vitreous fluid in the origin of such serious diseases of the eye as opacities of the lens and glaucoma. Many secrets still lie hidden in the vitreous.

I. Construction and Composition of the Vitreous

After the cornea has been abscised and the iris and lens removed from the eye of an animal, the fossa patellaris, in which lies the posterior surface of the lens, is laid bare.

The vitreous is an ectodermal formation, both anatomically and embryologically. It consists of a framework of fine fibres, part of which are radiating, part concentric. Nothing of this framework can be seen with the naked eye. Between the retina and the vitreous is a delicate limiting membrane, which was formerly known as the hyaloid membrane, but modern investigation has shown that it belongs to the limitans interna of the retina, for the framework of the vitreous comes from the glia elements of the retina. The fibres of the vitreous are densest and thickest in the neighborhood of the ciliary body, and the lamellar layers of this limiting membrane run in the immediate vicinity of the retinal limitans interna.

The entire large, remaining bulk of the vitreous consists of a gelatinous mass, which is perfectly clear and transparent. The great excess of the quantity of the vitreous fluid over that of the framework may be shown by passing the vitreous through a filter, when all of the fluid will pass through, leaving behind only a very small quantity of the substance of the framework. The composition of the vitreous fluid is quite similar to that of the aqueous.

It is important to know, with regard to the metabolism of this peculiar tissue, that it contains no lymphatics. It was formerly thought that a hyaloid canal continued to exist between the posterior pole of the lens and the papilla after the involution of the hyaloid artery, and to serve as a lymph passage, but this theory is not correct. The processes of metabolism in the vitreous will be described when we come to speak of the change of fluid in the eye, but I wish to say, with reference to the treatment of opacities of the vitreous, that very little effect can be produced on them by drugs introduced into the body.

II. Opacities of the Vitreous

1. *Muscæ Volitantes in the Healthy Eye*

This patient, who is slightly myopic, complains that when he looks at the bright sky, or at a white paper, little opacities float about in or in front of his eyes. They seem to him to be composed of round points, like strings of pearls, or to be irregular shadows. He says that he has often grasped for them, thinking that they were really flying hither and thither in front of his eye. He calls them midges; they are the so called muscæ volitantes.

In order to determine whether these opacities appertain to the vitreous we ask the patient whether they sink and continue to move when he holds his eye still. If the opacities constantly change their position when the eye is quiet they must be situated in the vitreous, for they could not do this if they were due to changes in the lens, but if we try to locate them with the ophthalmoscope we shall search for them in vain.

Opacities of the vitreous and of the lens are examined best with an ophthalmoscope having a strong plus lens in the aperture (*Lupenspiegel*), as in this way they are made to appear enlarged and more distinct, but even with this means we are unable, as a rule, to detect minute opacities in the vitreous. The patient alone is able to observe them through entoptic perception of the little shadows they cast on the retina. These muscæ volitantes are seen most distinctly by the myopic eye, and therefore myopes are the most apt to complain of them, although they are present in other healthy eyes.

The conditions that account for these muscæ volitantes are remnants of embryonal cells and fibres. Single wandering leucocytes also may stay for a long time in the vitreous. This form of muscæ volitantes is perfectly harmless and can cause trouble only when they are exactly in the region of the visual line. The phenomenon may often be observed while using the microscope. Muscæ volitantes are a cause of complaint with many nervous people, whose apprehensions we may relieve completely; they are helped most when they are taught to pay no more attention to the opacities.

2. *Opacities of the Vitreous in High Myopia*

The opacities in the vitreous of our next case, a patient with high myopia, are to be estimated quite differently.

One of the results of the dilatation of the posterior segment of the eyeball in myopia is that the nutrition of the posterior portion of the vitreous is impaired, so that both the gelatinous mass and the vitreous fibres become more fluid. This condition is known as fluidity of the vitreous, or as synchysis. Some of the fibres are ruptured mechanically by the dilatation, others become disorganized. Spaces filled with fluid are formed in the jelly, and opacities, that can be seen readily with the ophthalmoscope, float about in this fluid vitreous. These opacities assume many shapes, like points, particles, whip lashes, coils, and threads. They are thrown about, here and there, by the movements of the eye, and we make use of this fact in locating them. We direct the patient to make some quick movements of the eye and then to hold it quiet; then the opacities sink slowly and pass through our field of vision. Diseases of the chorioid and retina play a considerable part in their production.

The fluidity of the vitreous may produce further consequences: the volume of the substance of the vitreous may decrease, and then the tension of the eye becomes reduced. For this reason we are always obliged to keep watch over the intraocular tension when we have to deal with this class of cases. It seems hardly necessary to call special attention to the fact that the retina may readily become detached in such a soft eye in which the vitreous has become pathologically changed.

The zonule also is softened and atrophic in many cases of fluidity of the vitreous, so that the lens becomes tremulous and may become completely luxated.

3. Synchysis Scintillans

A change that is occasionally met with in the otherwise healthy eyes of old people was found in this patient after extraction of a cataractous lens. A large number of spangles that have a golden lustre may be seen to dance about in the vitreous; if the patient holds his eye still they sink to the bottom of the chamber like a rain of gold. This wonderful spectacle is caused by numerous crystals, composed of cholesterin, tyrosin and margarin, that reflect the light strongly. The origin of these crystals and the conditions under which they are excreted into the vitreous are still unknown.

4. Inflammatory Opacities in the Vitreous

The commonest cause of opacities in the vitreous is an inflammation of one of the surrounding membranes. We find them in this case of recurrent iritis, and we have already learned that the exudate in iridocyclitis is likewise deposited in this tissue.

These vitreous opacities consist of cellular and fibrinous exudates, which originate chiefly from the ciliary body, but also come from the chorioid and retina; consequently we must search carefully for diseases of these organs when opacities have been found within the vitreous. Not infrequently the cause is to be found in an inflammatory focus in the peripheral portion of the chorioid. These inflammatory opacities may be situated in different parts of the vitreous, and may differ in their development in accordance with the nature and severity of the intraocular disease. In some cases the vitreous may seem to be completely filled with a fine dust, in others the exudates are flocculent, and in still others they may adhere together and form dense membranes which hang down like sails and interfere materially with vision.

When the opacities are so extremely minute that they cannot be seen individually with the ophthalmoscope, the fundus appears to be uniformly hazy, to a degree proportional to the number of the opacities, but as long as the fundus can be seen one phenomenon is always striking: the papilla seems to be redder than usual, because it forms a bright background behind a cloudy medium, just as the sun looks like a red ball of fire on a foggy morning. When such a papilla is seen through a cloudy vitreous it is not always easy to determine whether a neuritis is present or not. When the opacity of the vitreous is still greater, only a reddish reflex may be obtained, or nothing at all can be perceived through the pupil.

As these inflammatory opacities of the vitreous are exudates, a part of them may undergo resorption and a part may become organized into connective tissue. Whenever the masses of exudate are of considerable size, blood vessels proliferate into them from the vessels of the retina.

Most of these inflammatory opacities of the vitreous are very refractory to treatment. The principal reason for this is the slowness of the intraocular metabolism, but it is partly due to the fact that the remedies employed arrive at the place where they are needed in insufficient concentration. The remedies ordinarily used are potassium iodide, or such modern preparations of iodine as sajodin, and mercury, in the form either of inunctions, or of pills. Subconjunctival injections of a 4% solution of salt have been much in favor recently. In suitable cases we may also try sweating with pilocarpin, wood tea (German Pharmacopœia), sodium salicylate, aspirin, or hot air baths. Abstraction of blood from the temple with *Heurteloup's* leech may be practiced, and footbaths may be ordered, but the therapeutic results obtained in opacities of the vitreous are very poor.

5. *Opacities of the Vitreous in Cataracta Incipiens*

This patient has an incipient senile cataract, in which the disease of the lens has begun, as it so often does, in the posterior cortex. Opacities in the vitreous are frequently found, when cases of incipient senile cataract are carefully examined. I do not believe this to be accidental. It seems to me to be more likely that the lens becomes diseased in such eyes because it lies for years in contact with an intraocular fluid which is pathological in its composition.

6. *Hemorrhages into the Vitreous*

The next case is one of hemorrhage into the vitreous due to a perforating wound. Hemorrhages into the vitreous may also be caused by contusions, and we meet with them in certain diseases of the eye, such as high myopia and chorioiditis, as well as in general diseases characterized by a pathological composition of the blood, such as chlorosis, anæmia, diseases of the blood vessels, and diabetes. In old people they frequently occur in connection with arteriosclerosis. The hemorrhages may come either from the blood vessels of the retina, chorioid, or ciliary body. In some cases the place of rupture of a retinal vessel may be seen with the ophthalmoscope.

Recurrent hemorrhage into the vitreous in otherwise healthy eyes forms a peculiar clinical picture, usually met with in young men, in whom, at irregular intervals, hemorrhages take place into the vitreous from the vessels of the retina. These intraocular hemorrhages frequently occur coincidently with nosebleed. Their nature is still unknown; more accurate investigations of the blood according to modern methods are urgently needed for its elucidation. In these cases it is very remarkable how rapidly the resorption of such a hemorrhage takes place sometimes, while that of an ordinary hemorrhage, due to other causes, is wretchedly slow and incomplete. If the hemorrhages recur frequently they may become organized into a peculiar white mass, which may also become vascularized. If such a mass lies in front of the retina the picture of retinitis proliferans may be presented. The hemorrhage may also lie between the retina and the vitreous, when it forms a peculiar ophthalmoscopic picture. A large, round bleb of blood is formed in the fundus, usually in front of the macula; after a while the blood sinks and the upper margin of the hemorrhage forms a horizontal line. These so called preretinal hemorrhages are due to various causes, but they usually occur in young persons. In rare cases the disc of blood lies in front of the papilla.

The fundamental principles of the treatment of hemorrhages into the vitreous are the same as those for the treatment of inflammatory opacities: rest, regulation of the diet, purgatives, sweating, and inunctions when the diseases of the vessels are syphilitic. Small hemorrhages may be resorbed quite well, but the process is very slow. The iris frequently becomes discolored in the course of the resorption, so that a blue or gray one appears of a greenish hue. Pigmentation of the retina may follow a hemorrhage into the vitreous.

Foreign bodies, such as spicules of metal, are frequently to be seen within the vitreous when the eye has been injured, but in order that the student may comprehend how such a particle of metal appears in comparison with other opacities, an opportunity should be furnished him to examine with the ophthalmoscope the eye of an animal into which a bit of iron has been artificially introduced.

7. *Parasites in the Vitreous*

The cysticercus, the larva of the tænia solium, which comes from the hog, is occasionally to be met with in the vitreous. Thanks to the hygienic measures carried out in the inspection of meat this parasite is now rarely seen. It enters the vitreous from the vessels of the chorioid or retina, becomes gradually encapsuled in connective tissue, and causes serious injury through the contraction and irritation it induces. Filariæ have attracted attention recently, because of the trouble they occasion people in our colonies. They likewise enter the vitreous and give rise to serious intraocular inflammation. I am able to exhibit such a filaria, which I removed from a gentleman employed in Camaroon, not indeed from the vitreous, but from the subcutaneous tissue of the upper lid, in which it had a lively, sinuous movement.

VIII

Diseases of the Sclera

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106. The Differential Diagnosis of Scleritis

Near the margin of the cornea of the right eye of this girl, about in the region of the insertion of the external rectus, is a livid spot over which the conjunctiva is distinctly elevated. The conjunctival vessels over it are injected, but other vessels, which have a violet appearance, shine through from the depths of the nodule. The prominence is larger than a lentil and blends with the surrounding tissue. It is exquisitely sensitive to the touch. A zone in which the sclera exhibits a peculiar livid discoloration almost surrounds the cornea.

The first question in such a case is whether the focus of disease is in the conjunctiva, or in the sclera.

The only thing this could be, if it were in the conjunctiva, is a solitary nodule of conjunctivitis eczematosa, but such a node can usually be differentiated by its appearance from a scleritic swelling; the injection is conjunctival, while in this case the color of the injection is bluish. The solitary efflorescence is also situated in the conjunctiva and may be moved with it here and there, while in this case it is impossible to move the firm nodule in the sclera. Furthermore, its surface becomes ulcerated very quickly in conjunctivitis eczematosa, but in this patient the nodule has existed for weeks and months, and it is a very characteristic feature of scleritis that these foci of inflammation never ulcerate. In doubtful cases the course will determine very soon whether the disease is conjunctivitis eczematosa, or scleritis.

Our patient presents still another characteristic: When the focus of inflammation in the sclera finally undergoes involution it is very apt to leave behind a peculiar, slate blue discoloration, while an eczematous nodule in the conjunctiva leaves no trace when it disappears. It was this peculiar discoloration that attracted attention when we began to examine the patient.

When we inquire further with what else this scleritic swelling could be confounded, the only suggestion is that of a large pinguecula. When the conjunctiva is inflamed a pinguecula may occasionally be distinctly elevated, but I do not think it could be mistaken for this condition. Hence we know that this is a disease of the sclera.

We must first study the normal sclera in order to understand this inflammation. The sclera is thickest near the optic nerve, gradually becomes thinner toward the equator, and is thickened somewhat anteriorly by the fusion with it of the tendons of the recti. It is constructed histologically of bundles of firm connective tissue and elastic fibres in a manner similar to that of the cornea. The bundles of connective tissue run in meridional and equatorial directions and interlace, with spaces containing flat cells between them. Pigment cells also are met with near the corneoscleral margin. This pigment can often be seen in the living eye, in the form of little brown spots on the white membrane, at the place where the anterior ciliary vessels emerge from the sclera. The fibres of the sclera pass over into those of the cornea, at the margin of the latter, quite gradually and imperceptibly, but in the cornea they are transparent and arranged much more regularly. The sclera itself has very few vessels; the main reason why diseases of this membrane are comparatively rare. Still, this is not a satisfactory explanation, for the cornea is just as destitute of vessels and yet it is frequently diseased. The agents of inflammation have difficulty in finding adequate conditions for development in the firm, almost tendinous tissue, and yet there are a number of general diseases in which the sclera is involved.

Let us study some sections of an eye with scleritis, in order to obtain an idea of what is taking place in the sclera of our patient. A very dense infiltration of round cells occupies almost the entire thickness of the membrane. The ciliary body is richly permeated with cells and has a fibrinous exudation on its surface. Lines of round cells follow the blood vessels toward the cornea from the focus of inflammation in the sclera, and the infiltration may be seen to extend even into the deeper layers of the cornea itself, where it is accompanied by numerous vessels. Now we see how far into the eye of our patient the effects of the inflammation of the sclera may have extended.

We make a clinical distinction between a superficial scleritis, also called episcleritis, and a deeper form, the true scleritis. This distinction is of clinical importance, although no sharp differentiation can be made between them pathologically, because experience has taught us that the prognosis of the superficial form is good, in that no serious injury to the interior of the eye is associated with it, while it is otherwise with the deep scleritis, which involves other portions of the eye.

In an episcleritis the layers of the sclera are forced apart and made to bulge by the exudate at the place where the nodule is situated. Such a node is obstinate, it may last for weeks, but finally it disappears through resorption and leaves behind either no changes, or a little slate gray spot in the sclera. This form of scleritis may recur frequently. After the disease has lasted for some weeks the first node disappears and a new one springs up in another place. This may be repeated until the disease has actually journeyed around the cornea, but it finally ceases, for it never appears a second time in the same situation.

These nodes likewise appear and undergo involution in the deeper and graver form of scleritis, but the sclera may be so thinned at the place of the deep infiltration as to give way before the intraocular pressure and to become ectatic. If the disease has also wandered around the cornea the entire gray zone may become broader; then the cornea moves forward, the eye is lengthened sagittally, becomes pyriform and highly myopic, or the sclera may bulge at certain places and allow staphylomata to develop. As these nodes are usually situated in the region of the ciliary body the final result of a grave scleritis may be a ciliary staphyloma.

Other changes still take place in the deep form of this disease. The inflammatory infiltrates are very apt to extend from the sclera into the cornea, as we have seen in the microscopical examination. The so called sclerosing keratitis then develops, which is thus an accompanying symptom of scleritis. We find an opacity in the deeper layers of the cornea extending from the scleral node at its margin, which possesses correspondingly deep vessels. This inflammation never produces ulceration of the cornea. Sclerosing keratitis has another unpleasant peculiarity: it is not very capable of involution, and in most cases the opacity is permanent. Its appearance changes to a tendinous, bluish white, and then it looks as if the sclera itself had extended into a portion of the cornea.

The fact that the scleritic swelling involves the ciliary body explains the changes that we meet with in the iris, an inflammation of which is present with all its symptoms, deposits on *Descemet's* membrane and posterior synechiæ. The involvement of the uvea is betrayed also by the fact that there are exudates in the vitreous. The other membranes of the eye are involved in this manner in a deep scleritis.

In scleritis posterior the nodes are situated farther back and are not in the neighborhood of the margin of the cornea. They are characterized by severe pain and a considerable swelling of the episcleral tissue at one canthus. The retina may be rendered cloudy and be bulged forward as by a tumor.

The differentiation, whether we have to deal in this case with a superficial, clinically benign episcleritis, or a deep, grave scleritis, is no longer difficult; we must not content ourselves with looking at and feeling of the node, but must make a careful examination of the entire eye. We find first a tonguelike, gray opacity at the outer margin of the cornea, situated in its posterior layers, as is shown by the deep vascularization. We find also quite a number of deposits on its posterior surface, which indicate the involvement of the iris and ciliary body in the form of a serous iritis.

Rheumatism and gout are the preeminent causes of this disease, but there is no doubt that in many cases it is due to a benign tuberculosis. It must be admitted that the etiology is frequently difficult to determine, even when the organism has been thoroughly examined with the aid of biological reactions. The fact that an inflammation of the sclera is usually very obstinate and requires a long time for involution is due largely to the anatomical peculiarities of this tissue, with its probably slow metabolism, and to the uncertainty of the etiology. We must therefore tell this patient that she must have patience, that the course of the disease is very slow. When there are signs of a concomitant iritis the eye is kept under the influence of atropin. Heat is applied constantly at first by means of the thermaphore. If the cause seems to be rheumatism we prescribe aspirin, and may also use preparations of iodine. When the node becomes less painful we try to accelerate its resorption by massage and dionin, but we cannot prevent recurrences. If signs of gout are present, treatment of the uric acid diathesis is indicated. In the course of gout we also meet with the so called episcleritis periodica fugax, in which discrete patches of infiltration of the episcleral tissue appear and soon disappear, but recur for a long time with a certain regularity. If the result of the tuberculin test shows that tuberculosis is the probable cause a course of tuberculin treatment may be instituted.

IX

Diseases of the Lacrymal Organs

107. The Normal Functions of the Lacrymal Organs

Our patient complains of an extremely characteristic symptom: his eyes are constantly full of tears. The lacrymation is not so great while he remains quietly in the house, but as soon as he goes out of doors and is exposed to the wind and weather it becomes so marked that tears run down his cheeks from time to time. This phenomenon developed gradually, has persisted for a long time and is very annoying. This overflow of tears, which we call epiphora, indicates the presence of something abnormal in the lacrymal apparatus of the eye. If we are to detect and obviate the cause we must be acquainted with: I, the anatomy of the lacrymal passages; II, the physiological function of the same; III, the most important causes of the overflow of tears; IV, the introduction of probes into the lacrymal passages and the treatment of strictures.

I. Anatomy of the Lacrymal Organs

The tears are secreted by the lacrymal gland, which lies beneath the margin of the upper and outer portion of the orbit, and consists of two parts of unequal size. The upper and larger portion, known as the orbital lacrymal gland, lies in a depression in the bony wall of the orbit, the fossa glandulæ lacrimalis. Its excretory ducts open in the outer part of the upper transition fold. The palpebral lacrymal gland, also called the accessory lacrymal gland, is smaller and is situated near the excretory ducts of the orbital gland, just above the upper transition fold, where it may be demonstrated in the living person. If we direct the patient to look downward, after the upper lid has been everted, we see the outer part of the upper transition fold pushed forward by a soft, yellowish mass, the palpebral lacrymal gland, but this gland is not to be reached so easily in every person, we often have to press the upper transition fold forward to bring it into view.

In addition to these principal ones minute glands of a similar character are scattered about in the conjunctiva, situated for the most part in the upper transition fold and known as *Krause's* glands. Similar little glands are also met with in the tarsus, above and below the *Meibomian* glands.

The lacrymal gland is constructed anatomically from an agglomeration of tubular glands, with numerous lymph cells in the connective tissue between the lobules. The adenoid tissue is absent in infants, according to *Axenfeld*, and as age advances the intermediate tissue increases, while the glandular tissue slowly retrogrades. The glandular tubes contain a single layer of cylindric cells, which represent the gland cells. I cannot go into the histological details of the glandular protoplasm, but will simply say that the quiescent cells contain very many granules, and that substances are normally met with in the gland cells which give the reaction of fat. Investigations have also shown that all cells do not work uniformly during the secretory activity of the lacrymal gland, but that many portions rest while other parts are actively engaged in secretion.

The tears secreted by the lacrymal gland are composed of a sterile, slightly opalescent, colorless fluid, as clear as water, and having an alkaline reaction. Under the microscope we find in it here and there some desquamated, epithelial cells. One hundred parts of lacrymal fluid contain, according to *Frerichs:*

	I	II
Water	99.06	98.70
Epithelium	0.14	0.32
Albumin	0.08	0.1
Mucus and fat	0.3	0.34
Sodium chloride and other salts	0.42	0.54

The salts contained are, in addition to sodium chloride, carbonates of sodium and of magnesium, sulphates and phosphates of lime. From the small amount of salts it contains the lacrymal fluid must be hypisotonic, but if we instill hypisotonic solutions into the conjunctival sac they prove to be just as unpleasant as hyperisotonic solutions, therefore, if it be granted that the analyses are correct, it remains to be proved whether the action of the hypisotonic lacrymal fluid is compensated for by dissolution in a colloidal fluid.

With regard to the bactericide action of the tears, it was proved, when speaking of the conjunctiva, that this frequently asserted bactericide action has been authenticated only with regard to certain kinds of bacteria, and is in reality to be ascribed to the fact that the lacrymal fluid forms a bad nutritive medium for many bacteria.

The lacrymal fluid passes from the mouths of the excretory ducts into the conjunctival sac, whence it is guided into the puncta lacrimalia, situated in the free margins of the upper and lower lids near their inner ends. Under normal conditions they are so situated that the upper one points somewhat backward and downward, the lower one somewhat backward and upward, so that the openings touch the eyeball and slide upon it as it moves. They are placed on little elevations, the lacrymal papillæ, which vary in size in different people and ordinarily are much elongated in old persons.

The puncta lacrimalia are the openings of the canaliculi, which run vertically at first, for about two millimetres, and then bend and run horizontally toward the inner canthus for eight or ten millimetres. They are lined with a laminated pavement epithelium and are surrounded by a dense network of elastic and muscle fibres. This fact, which is of considerable importance in its physiological work, has been made a certainty by modern research, especially by *Halben.* The muscle fibres are arranged circularly about the vertical portion of the canaliculus and run parallel to the horizontal portion.

The canaliculi converge inward and finally open into the lacrymal sac, either close together, or united in a short, common trunk. The opening corresponds to about the middle of the internal tarsal ligament and presents, in many cases, an anatomical construction like that of the lacrymal sac.

The lacrymal sac, together with the nasolacrymal duct, forms the true lacrymal duct. The lacrymal sac is from 10 to 14 mm long, the nasolacrymal duct from 15 to 20 mm. The lacrymal sac is situated at the inner canthus in the fossa sacci lacrimalis of the lacrymal bone, so that it rests securely upon the bones behind and to its nasal side, while it is covered in front and to the temporal side by the ligamentum canthi internum and the periorbital membrane.

In order to locate the lacrymal sac in the living person we draw the lids toward the outer canthus so as to cause the ligament of the internal canthus to project somewhat. The lacrymal sac lies behind this ligament in such a way that its apex extends somewhat above the upper margin of the latter.

It is necessary to study microscopically the structure of the walls of this organ in order to understand its diseases. Within we find several layers of cylindric epithelium which rest on a structureless basal membrane. The mucosa consists of a loose, reticulated connective tissue with elastic fibres and so many lymphoid cells that follicles may be formed in catarrhal irritations. The submucosa consists of a firm, fibrous tissue which contains such a strikingly large number of elastic fibres that they must be of great importance in the guidance of the tears. In rare cases small glands are also present in the mucous membrane of the lacrymal sac.

The lacrymal fluid passes from the lacrymal sac into the ductus nasolacrimalis, the lining of which is of similar construction to that of the walls of the sac: laminated cylindric epithelium, mucosa with numerous lymph cells and occasionally containing follicles, submucosa with a rich venous plexus. The nasolacrymal duct finally empties below the inferior turbinate into the nose. The submucous venous plexus is very much developed about its mouth, but the principal thing that differentiates this portion of the lacrymal passage from the lacrymal sac is the fact that it is surrounded on all sides by bone, that it is embedded in bone. The importance of this in pathology will soon be evident, but we must first learn the physiology of the lacrymal organs.

II. Physiological Functions of the Lacrymal Organs

A. Function of the Lacrymal Gland

Our views concerning the means by which the lacrymal gland is caused to secrete have undergone a change. We have known for a long time that the lacrymal nerve, which comes from the ophthalmic nerve, the first branch of the trigeminus, supplies the lacrymal gland. Its upper branch passes through the gland and is distributed in the skin and conjunctiva about the outer canthus; its lower branch unites with the temporal zygomatic nerve, which comes from the second branch of the trigeminus. The lacrymal gland is also supplied by fibres from the sympathetic, which accompany its nutrient vessels.

Until within a comparatively short time the trigeminus was supposed to be the secretory nerve of the lacrymal gland, and numerous experiments were made to trace this secretory nerve more accurately by means of stimulations and reactions, but when *Goldzieher* showed in 1893 that the lacrymal secretion may cease in facial paralysis, and that therefore the facial must be the secretory nerve of the lacrymal gland, the belief in the secretory importance of the trigeminus was shaken. We know now, from the labors of *Koster*, and of others, that the secretory nerve of the lacrymal gland really comes from the facial. It has been proved that the secretion of the lacrymal fluid is disturbed in facial paresis if the lesion is situated in the region of the ganglion geniculi, whence the conclusion may be drawn that the secretory nerves for the lacrymal gland must leave the facial at some point in its course through the petrous portion of the temporal bone. The clinical observations of *Koster* in cases of facial paresis have shown that these fibres are no longer present in the trunk of the facial nerve near the ganglion geniculi, so it is quite probable that they pass through the nervus petrosus superficialis major to the sphenopalatine ganglion, thence to the second branch of the trigeminus, and then through either the nervus subcutaneus malæ to the gland, or back to the *Gasserian* ganglion and thence by way of the first branch of the trigeminus and the lacrymal nerve to the gland. From what source they come, and whether they originate in the facial nucleus itself, is uncertain. The lacrymal secretion is not stopped in congenital facial paralysis in which the nucleus of the facial nerve is absent or imperfect. This indicates that the secretory fibres for the lacrymal gland do not come from the facial nucleus itself, but from some other nuclear region, probably from that of the glossopharyngeus, which also furnishes the nerves for the salivary glands, and pass from this place to join the root of the facial nerve, probably through the portio intermedia *Wrisbergii.* It is probable, though not yet proved with certainty, that, in addition to these secretory nerves, the lacrymal gland is supplied with fibres of the sympathetic that possess secretory power, which enter by way of the vessels. In weeping, the vessels of the face and conjunctiva dilate, and we may suppose that this vascular dilatation extends to the lacrymal gland. The sympathetic has therefore been considered by not a few to be the secretory nerve of the lacrymal gland. It may be that the lacrymal gland has a double supply of secretory nerves, the same as the salivary glands. I believe, with *Arloing* and *Laffay,* that fibres inhibitive of secretion are present in the sympathetic.

However this may be, the lacrymal gland may be excited to secretion in two different ways, through direct innervation from a centre unknown as yet that produces psychical weeping, and by reflex action from the trigeminus or the optic nerve.

We do not know with certainty the situation of the centre for psychical weeping, which is excited by the emotions. It may be in the optic thalamus which, according to *Nothnagel,* is the seat of the mimical movements of expression. Psychical weeping is peculiar to man, none of the lower animals, not even one of the anthropoid apes, have this reaction to the emotions. It is a matter of interest in this connection that an infant does not weep psychically, and still its lacrymal gland is well developed, although it does not yet contain as much adenoid tissue as an adult's. It can also be easily demonstrated that the lacrymal gland of the new born infant secretes as the result of reflex irritation, so, as it cannot respond to psychic stimuli, we must suppose that certain tracts in the brain do not yet perform their duties. Every mother knows that the first tears do not come until the child is several weeks old. It is also well known that men weep less than women. In man a hyperplasia of the stroma takes place, together with an atrophy of the glandular tissue. A lacrymation is also associated with yawning, laughing, coughing and vomiting, which is probably due to an innervation extended to the lacrymal gland. Psychical weeping is of interest to us just now only because in it may be perceived the involvement of the sympathetic. In weeping the conjunctiva reddens, then the margins of the lids, the lids, and finally the face swell and redden; all results of the dilatation of the blood vessels.

The secretion of the lacrymal gland may be obtained not only by psychical weeping, but by reflex action. Thus the eyes lacrymate when we look at the sun, or are dazzled by a bright light. Sensitiveness in this respect varies a great deal. The reflex tract followed in such a case is by way of the optic nerve.

Lacrymation from stimulation of the trigeminus is of still greater clinical interest. Such a reflex action is to be met with almost daily in connection with diseases of the eye. Conjunctivitis, keratitis, iritis, may excite an increased secretion of the lacrymal fluid through irritation of the fibres of the trigeminus.

The commencing dryness of the surface of the eye, the agitated air, and the increased concentration produced by evaporation constantly govern the secretion of the lacrymal fluid in a reflex manner, which ceases only during sleep. This reflex lacrymation is therefore the secretion of the lacrymal gland that is maintained continually during the waking hours of the day.

When we extirpate the lacrymal gland the eye does not become dry, because its moisture is guaranteed by the conjunctiva alone. We might suppose that, under normal conditions, this duty rested upon the conjunctiva alone, and that the lacrymal gland performed its function only when stronger irritations affect the eye, but *Schirmer* has shown that in persons whose lacrymal sacs have been extirpated, but otherwise have unirritated eyes, more tears are constantly secreted than are evaporated from the palpebral fissure.

Attempts have been made to measure the quantity of fluid secreted in this reflex manner. Weighed strips of filter paper have been placed in the conjunctival sac in order to determine from their increase in weight by the moisture, the quantity of lacrymal fluid produced in a given time. These showed that from 0.5 to 0.75 gram of fluid is secreted in sixteen hours, but eyes from which the lacrymal sac has been extirpated are not physiologically normal eyes, particularly with reference to the irritability of the conjunctiva. When the eyes are perfectly normal a comparison of the two, tested in this manner, shows a considerable difference, and therefore we can hardly utilize such a comparison when a functional disturbance of one lacrymal gland is suspected. The extent and rapidity with which these strips of filter paper become wet before and after cocainization of the eye vary considerably. Even though this method is inexact and of no scientific importance or usefulness, we must still suppose, with *Schirmer*, that the lacrymal gland, under normal conditions, continually secretes a larger quantity of fluid than is disposed of by evaporation through the palpebral fissure. Therefore the tears must be regularly conducted toward the nose, and much more still must be demanded of the abducent passages when the amount of secretion is increased.

B. Conduction of the Tears

Various theories have been advanced to account for the mechanism of the conduction of the tears. After they leave the excretory ducts of the lacrymal gland they must spread over the entire conjunctival sac, if they are to keep the eyeball moist. This is accomplished primarily by the elastic pressure of the eyelids, which guides them into the palpebral fissure, where they sink downward on the eyeball, partly because of the attraction of gravitation, and accumulate in the middle of the intermarginal portion of the lower lid. Capillary attraction also takes part in the covering of the eyeball with a layer of fluid. In this manner the tears accumulate along the intermarginal portion of the lower lid and in the lacus lacrimalis, where the puncta lacrimalia are immersed.

Weber called attention to the fact that the lacrymal tube resembles a siphon, the shorter branch of which is formed by the canaliculus, the longer one by the lacrymal sac and the nasolacrymal duct, and therefore it has been widely supposed that the tears were removed by siphonage, but more modern researches, particularly those of *Schirmer*, have shown that when the lids are not moved the tears are not carried to the nose, so the siphon does not always work.

It was formerly thought by *Molinelli* that capillary attraction took part in the conduction, as it was still believed at that time that the canaliculi were filled with air from time to time, but it is known now that normally the lacrymal duct is always filled with fluid.

According to *Honnauld* the tears are aspirated into the nose on inspiration; the current of air in the nose sucks the fluid from the nasolacrymal duct, but in *Schirmer's* experiment no easily demonstrable bacteria reached the nose, even when the respiration was purely nasal, when winking was discontinued. Thus the importance of the movements of the lids in the conduction of the tears is continually brought to our attention.

The lids are capable of three movements: a rapid opening and closure, as in nictitation, a snapping of the lids together, as in winking, and the closure of the eye, and the question arises: Which movement has the greater power to drive the tears along?

It was believed by *v. Arlt* that the lacrymal sac was compressed when the lids were closed, and that the fluid contained in it was driven into the nose, but this is fundamentally incorrect, because there is no muscle connected with the eye that could produce such compression. When the orbicularis contracts it enables the ligamentum canthi internum to dilate the lacrymal sac. It is a matter of common observation that tears standing in the eye can be conducted into the nose best by quickly repeated snappings of the lids together, and more recent studies have shown that the driving power in the conduction of the tears is not the closure of the lids, but the slightly twitching motion they make in winking. *Schirmer* claims that this is the only tear conducting force.

In order to understand the conduction of the tears we must conceive that in winking the contracting portion of the orbicularis draws the ligamentum canthi internum somewhat forward and outward, so that, as the outer wall of the lacrymal sac is firmly attached to the ligament while its nasal wall is fixed by the periosteum, the lumen of the sac is increased somewhat by the act of winking and thus the lacrymal fluid is sucked into the lacrymal sac. As soon as the contraction of the orbicularis relaxes, the walls of the sac are drawn together again by their elasticity.

The purpose of the elastic tissue in the wall of the lacrymal sac is evidently to afford the necessary contraction when the sac is dilated. The anterior wall is strengthened in this respect by the ligamentum canthi internum; it is rather deficient in elastic tissue in only one place, and it is there that ectasiæ form in chronic disease of the sac. The tears are pressed by the elastic compression out of the lacrymal sac through the nasolacrymal duct, because this duct is larger than the canaliculi. Probably the muscles and elastic tissue about the latter also act to prevent a reflux of the fluid into the conjunctival sac. The anatomical condition of the elastic and muscular tissue about the canaliculus renders it probable that a sort of sphincteric action is present in its vertical portion, where a contraction is frequently felt to take place after a knife has been introduced for the purpose of slitting the canaliculus.

When the lids are spasmodically closed a great quantity of lacrymal fluid accumulates without flowing away. The probable explanation of this is that the upper portion of the canaliculus is occluded by these little sphincters. Both the muscular and the elastic tissue act to dilate the lumen of the canaliculus in its horizontal portion. The conduction of the tears is by suction into the lacrymal sac, by its dilatation through muscular contraction, and then by being sent farther along by its contraction. This mechanism performs an important duty, for the eye must be maintained in a certain degree of moisture if the cornea is to remain transparent.

The conjunctiva itself furnishes only a very small portion of the fluid, except when the lacrymal gland has been removed or has lost its function; under normal conditions it has a different duty to perform. Processes of diffusion continually take place between its tissue and the fluid in its sac by which an increase in the concentration of the fluid is prevented. A portion of the fluid evaporates, but the greater part is constantly guided toward the nose. The process is very slow. *Schirmer* has calculated from the time taken by bacteria coming from the conjunctiva to arrive in the nose that the rapidity of the current in the lacrymal passage is less than one millimetre per minute. The mechanism for the removal of the tears is so regulated that it is equal to greater demands. The slowness of the current under normal conditions is explained by the slowness of the supply. Thus four factors cooperate in the regulation of the normal change of fluid in the conjunctival sac; lacrymal gland and conjunctiva, evaporation and conduction of tears. So much for the theory of the normal function of the lacrymal apparatus.

We suspect that the outflow of tears from one eye of this patient is obstructed, but how is it to be proved? All that is necessary is to instill a few drops of a salt solution stained with fluorescin, or of a 1% solution of fluorescin, into the conjunctival sac and then direct the patient to wink rapidly a few times. If the lacrymal passage is free the fluorescin will appear in the nose in a few minutes and can be easily demonstrated by having the patient blow his nose. The coloring matter is demonstrable only on the healthy side of our patient. Far too little use is made in practice of this simple method. Whenever, in the course of a chronic conjunctivitis, the suspicion arises that the passage of the tears is obstructed this test should be applied.

108. The Most Frequent Causes of Epiphora and the Introduction of Probes into the Lacrymal Passages

I. The Most Frequent Causes of Epiphora

Epiphora is a symptom that may be due to three different causes: hypersecretion of the lacrymal gland, absence of driving power in the conduction of the tears, and obstruction of the lacrymal passages.

A hypersecretion of the lacrymal gland may be so great that the apparatus for conducting away the tears is not able to cope with the demand, as in psychical weeping, when there is a direct innervation of the gland. Such weeping is met with in hysterical persons who complain of the tendency to weep. Reflex irritation of the lacrymal gland also takes part in the increased flow of tears in exophthalmic goitre and in migraine, as well as in the rare cases in which an increased secretion is met with in cases of tabes and as a reflex symptom in pregnancy. Above all it is a symptom of the most various irritations and inflammations of the eye, such as foreign bodies in the conjunctival sac, or keratoconjunctivitis eczematosa. We have to determine first whether such a reflex irritation of the lacrymal gland is produced by disease of the conjunctiva or cornea in every case of epiphora. These factors are excluded in this patient, who complains of a watery eye.

The next thing to be determined is whether the epiphora is due to absence of driving power in the conduction of the tears. We meet with such a condition in facial paresis. The movement of the lid in winking is the only driving element in the conduction of the tears, and this is absent when the orbicularis is paretic. The secretion of the lacrymal gland fails in many cases of facial paresis, because its innervation is paralyzed, but in such cases the consequent lagophthalmos very quickly produces a condition of irritation in the conjunctiva which causes an increased secretion on the part of this membrane, and, as the tears cannot be carried away, epiphora is produced, even when the branch of the facial that innervates the lacrymal gland is paralyzed, when the lesion is in or above the geniculate ganglion. Even when there are no other conditions of irritation of the eye the little fluid secreted by the conjunctiva suffices to form an accumulation along the lower lid, although it is not usually enough to overflow upon the cheek. Incomplete coaptation of the lids may also occur, due to a shortening, or a notching of them, or in rare cases to a hypertrophy of the plica. None of these factors exist in the present case.

When old people complain of epiphora the usual cause is an obstruction somewhere in the lacrymal passage. It may be situated in the puncta. Normally the puncta are visible only when the lid is drawn out somewhat from the eyeball, but in old people the papillæ, in which they are situated, may be elongated so that the puncta are no longer in apposition with the surface of the eyeball, but are directed upward or downward, dip no longer into the lacus lacrimalis, and thus the conduction of the tears is interrupted. It is also possible for the puncta to be stenosed, or obliterated.

The canaliculus must be slit in all such cases. The margins of the wound must be exposed to the air for a few days, until they have become covered with epithelium, in order to prevent their adhesion. I am apt to combine with the slitting of the canaliculus the excision of its posterior wall as recommended by *v. Hofmann.* The wall is seized with forceps and cut away with scissors, thus forming a larger groove, from which experience has taught us a better conduction of the tears may be obtained.

Very often the puncta do not dip properly in the lacus lacrimalis because of an ectropion of the lids, due to blepharitis, conjunctivitis, or other causes; when this condition exists the faulty position of the margin of the lid must be corrected first.

The canaliculi may be obstructed in rare cases by concrements formed by proliferations of streptothrix. They may be distended in places by these deposits, and the walls may become inflamed. In such cases the canaliculi are to be slit and the concrements removed.

The obstruction is ordinarily situated in the nasolacrymal duct, either at the commencement of the bony canal, or at its opening into the nose. Strictures form at these places which interfere with, and often absolutely prohibit, the passage of the tears. The formation of such strictures is favored by the narrowness of the canal, and also by its anatomic structure.

The causes of these strictures are to be found chiefly in the diseases of the nasal mucous membrane, which are apt to extend to the mucous membrane that lines the nasolacrymal duct. The nasal diseases that are principally responsible for them are hypertrophic and atrophic rhinitis, but syphilitic and tuberculous ulcerations and inflammations may also produce them, as well as, occasionally, polyps and injuries of the nose. Hence the symptom of epiphora may be due etiologically to a disease of the nasal mucous membrane. Some of these cases need to be turned over to the rhinologist for treatment, but we can treat the larger number of them. Our duty is to cure the stricture and restore the way for the passage of the tears. In order to ascertain whether our suspicion is correct, that in this case there is stenosis of the nasolacrymal duct, we test the duct to learn whether fluid can pass through it.

I will illustrate this procedure first on a healthy person: The lower canaliculus is dilated with a suitable probe, the needle of an *Anel's* syringe is introduced into the lacrymal sac and then a salt solution, stained with fluorescin, is carefully injected. The patient bends his head forward and the fluid runs out of his nose. This experiment may succeed even when the sac is diseased and a stricture is present, for the injection of fluid under such a pressure may render the passage permeable for the moment, and it still needs to be proven whether the passage is open under physiological conditions. To make this test we instill a solution of fluorescin in the conjunctival sac, the patient winks a number of times and then, under normal conditions, after a few minutes the stain appears in the nose. Nothing of the sort takes place in our patient, and this makes the diagnosis of stricture almost certain, but for absolute proof we have to introduce a probe.

II. Introduction of Probes and Treatment of Strictures of the Nasolacrymal Duct

Probing of the lacrymal duct must be performed with the greatest care, with a gentle hand, and all violence that might lead to the creation of a false passage must be avoided. Some cocain is first instilled into the conjunctival sac, then the punctum is dilated with a small, blunt needle, if it is too small to admit of the entrance of the probe pointed canaliculus knife, a little cocain is injected into the canaliculus by means of a small syringe, and then the canaliculus is slit. In many cases a probe may be passed without a previous slitting of the canaliculus, but it is passed more easily after this has been done. The upper canaliculus may be slit for cosmetic reasons, especially in women, but usually the operation is performed on the lower one. If the right eye is to be probed, we stand behind the patient; if the left, we stand in front of him.

We first draw the lower lid outward and downward with the thumb of the left hand, so as to make the punctum easily accessible. We introduce the probe point of a *Weber's* knife vertically into the vertical portion of the canaliculus, bring the knife to a horizontal position, so it can be passed along the horizontal portion toward the nose, keeping its edge directed upward and somewhat backward so as to cause the incision to be situated toward the inner edge of the lid, and then push it forward until it strikes bone, when we know that it has reached the nasal wall of the lacrymal sac. As a precautionary measure we draw the knife back and forth a few times so as to be sure that its point moves freely in the sac and has not driven a fold of the mucous membrane before it. Then the handle of the knife is raised until it is in a vertical position and the canaliculus is slit. The length of the incision varies: it should be for the entire length of the canaliculus if it is desired to excise the posterior wall, as recommended by *v. Hofmann.* Probing also is easier when the division is complete. We next inject a few drops of a 2% solution of cocain, with some adrenalin added, into the lacrymal sac and introduce the probe.

Bowman's probes are made in various sizes. We take first a small one,

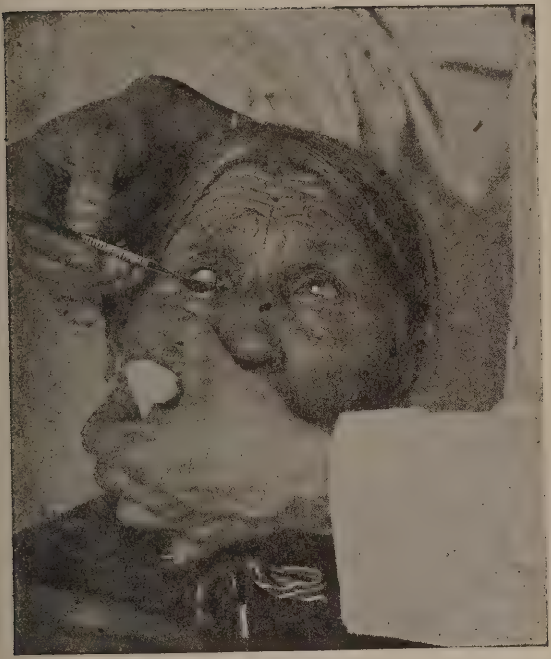

FIG. 81a

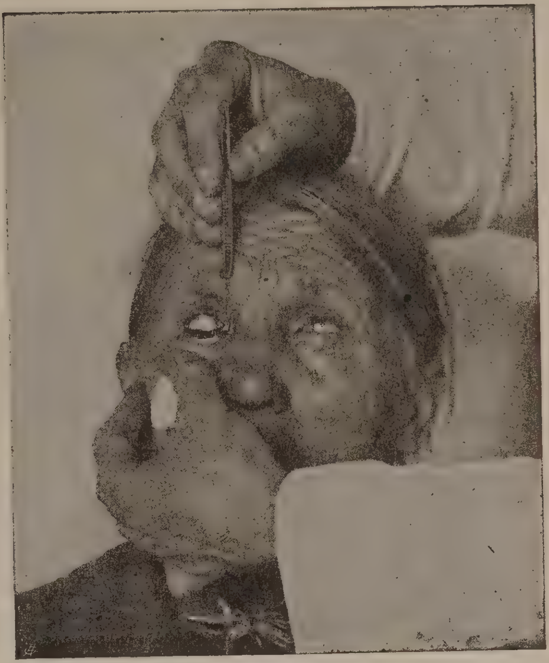

FIG. 81b

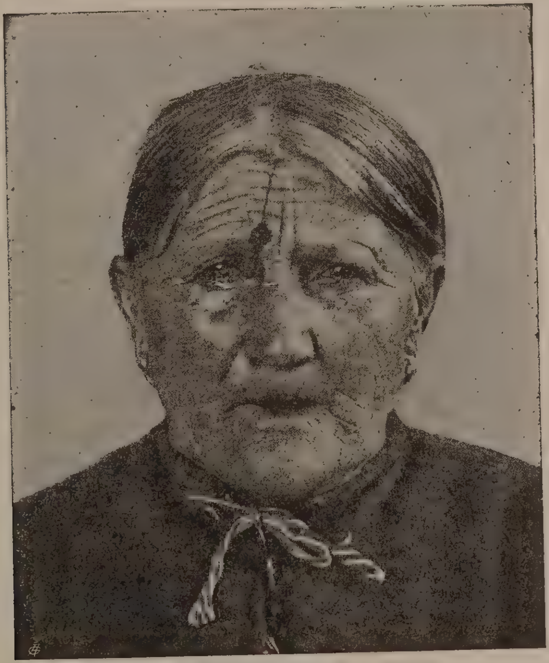

FIG. 81c.

Slitting the Canaliculus and Probing the Lacrymal Duct.

introduce it in the same way as the knife until it impinges on the bone at the nasal side of the sac, while the lower lid is tensely stretched outward. When the probe has entered well into the lacrymal sac it is raised to an upright position with its concavity forward, so that its point is directed to the angle between the wing of the nose and the cheek, before we begin to pass it down through the sac into the nasolacrymal duct. The two passages diverge downward. Now we pass the probe very carefully down into the nose and have overcome some strictures. The resistance of the suspected stricture can be felt in this case at the entrance of the bony canal and also at its lower end. Usually the patient states when he feels the probe in the nose, but we can perceive when this end has been attained by the position of the probe, with its plate lying on the brow. If the attempt to pass the probe is not successful no force should be employed, but another attempt may be made in a few days. Every attempt will fail when the stricture is bony and cannot be passed.

Unfortunately, strictures that can be overcome reform very quickly, so we have to repeat the probing every few days, and to try to pass a larger one each time so as to dilate the stricture. The probe should be left in position for several minutes.

If the patient cannot come with sufficient regularity for this treatment, but yet can remain under the care of a physician, we introduce a style for the purpose of securing dilatation. Styles may be left in place for several weeks, under certain circumstances; they usually become loose and fall out when the patient is washing. There is no doubt that this form of treatment with probes may cure both the epiphora and the strictures, but the percentage is very small in which permanent relief is given—my estimate is from 5 to 10%. More often there is a recurrence after a while, or there is no improvement at all. This is due to the nature of the fundamental disease in the nose. Probing is painful, in spite of cocainization, and little liked by patients, but if a stricture is allowed to persist chronic dacryocystitis develops. If we have to deal with a continually recurring tumefaction in the nose the inferior turbinate may sometimes have to be resected. When the epiphora occasioned by a bony stricture without suppuration of the lacrymal sac is very troublesome we may help the patient by extirpation of the palpebral lacrymal gland.

109. Chronic Catarrhal Dacryocystitis and Extirpation of the Lacrymal Sac

I am able to demonstrate by this patient the clinical picture that appears in a later stage of such a case of stenosis of the nasolacrymal duct as we have just seen. This woman complains of epiphora that is particularly marked when she is exposed to the wind and weather, that she has had pus in her eyes for a long time, that her lids are stuck together in the morning and that her eyes burn, especially in the evening. These symptoms are indicative of a chronic conjunctival catarrh, but something else is also present. The epiphora awakes a suspicion of disease in the lacrymal organs, and we find a swelling as large as a bean at the inner canthus of one eye, in the region of the lacrymal sac. The skin over the swelling is perfectly normal and freely movable. The swelling itself is not sensitive to pressure, but it becomes smaller when pressure is made upon it, while a mucopurulent secretion exudes from the puncta into the conjunctival sac.

I. Diagnosis of Chronic Catarrhal Dacryocystitis

This leaves no doubt that we have to deal with an inflammation of the mucous membrane of the lacrymal sac, and we have thus far learned these facts in regard to diseases of the lacrymal duct:

1. No such inflammatory secretion of the mucous membrane need be present in a simple stricture of the lacrymal duct, even when it is situated in the nasolacrymal duct. The symptom of stricture is epiphora, but its presence can be demonstrated only by means of a probe.

2. When the mucous membrane of the lacrymal sac becomes inflamed a pathological secretion is formed which can be pressed out of the sac by the finger. The finger must instinctively seek the lacrymal sac as soon as suspicion of a dacryocystitis arises, and such a suspicion must be excited whenever we meet with a chronic conjunctivitis of one eye alone, for which no other satisfactory explanation can be found. The purulent secretion that constantly escapes from such a lacrymal sac irritates the conjunctiva and maintains the inflammation.

3. The course of a chronic catarrhal dacryocystitis does not end in the formation of a secretion. The diseased lacrymal sac may enlarge considerably, even to the size of a walnut, and when such an ectasia of the sac is present the chronic dacryocystitis may be diagnosed from its appearance, even without the pressure of the finger. The diagnosis is extremely easy in every case, yet how often is the disease overlooked!

II. Etiology and Course of Chronic Catarrhal Dacryocystitis

I always emphasize the designation chronic catarrhal inflammation. This dacryocystitis is an extremely chronic disease which develops insidiously and may last for decades under certain circumstances. Its usual origin is as follows: The normal conjunctival sac contains infectious germs and pus agents which ordinarily are carried by the tears into the nose without trouble, but when a stricture has been formed in the nasolacrymal duct not only are the tears retained in the lacrymal sac, but an ever increasing number of pus agents accumulate in it. The patient has a simple epiphora and for a long time the pus agents die off, until finally a larger number, or more virulent ones, gain a foothold. Then they begin to multiply, to settle in the mucous membrane and the inflammation of the latter has begun. The mucous, or purulent, secretion, if examined in this stage, is found to contain numerous pneumococci, staphylococci, streptococci and other inflammatory agents. Microscopically the mucous membrane of the lacrymal sac appears to be thickened and infiltrated with pus cells.

At the height of the disease the eye lacrymates, the conjunctiva is in a constant state of catarrhal irritation, and large quantities of pus are present in the lacrymal sac. The patients notice their condition very soon and are accustomed to press out the contents of the lacrymal sac and to wipe out the eye, but the pus usually reaccumulates in a very short time. The disease may remain in this stage for a long time until some day an acute phlegmonous inflammation of the lacrymal sac and of the surrounding cellular tissue sets in.

If this does not happen the lacrymal sac goes on suppurating incessantly and the mucous membrane gradually becomes atrophic. This is perceived clinically from the fact that the secretion gradually loses its purulent character and becomes more mucous, and then thin fluid. The form of the sac also is changed. Its continuous distention, together with the atrophy of the mucous membrane, results in the gradual loss of elasticity by the wall. The lacrymal sac becomes more and more distended until its anterior wall, in which the elastic elements are less developed, gives way. The enlarged sac causes the skin that covers it to bulge forward, and forms an elastic tumor beneath the ligament of the inner canthus, its wall thickened, its elasticity lost. This condition is known as ectasia and atony of the lacrymal sac. Even if the patient still delays, the increasing size of the tumor will finally lead him to the physician.

Usually when we press upon such an ectatic lacrymal sac its contents will be emptied into the conjunctival sac, but it sometimes happens that they are evacuated into the nose, and cases have in fact been met with in which the outlet was obstructed in both directions, so that the contents could not be evacuated.

We have assumed that strictures in the nasolacrymal duct necessarily precede the development of such a dacryocystitis, but I do not question that it may originate in another way. The inflammatory agents coming from the conjunctiva may obtain a foothold in the epithelium without any stricture being present, and therefore a purulent catarrh of the mucous membrane may be present even when the passage is still free. The irrigating fluid does not meet with the least obstruction in such cases. The passage may remain open for a long time and the stricture may finally be not the cause, but the result of the dacryocystitis. The ordinary pus agents are the ones that are usually found, but in rare cases tuberculosis has established itself in this manner in the mucous membrane of the lacrymal sac.

III. Treatment of Dacryocystitis and Extirpation of the Lacrymal Sac

The treatment of chronic catarrhal dacryocystitis is extirpation of the lacrymal sac. The question may be raised why such a radical procedure should be adopted. Entirely aside from the troublesome epiphora, the continual irritation of the conjunctiva, the constant purulent condition of the eye maintained by the dacryocystitis, and without reference to the fact that such people are apt finally to develop a serious cellulitis, which alone is enough to render the timely abolition of the disease desirable, the lacrymal sac should be extirpated because of the relations of chronic catarrhal dacryocystitis to ulcus serpens, which destroys thousands of eyes every year. As long as the epithelium of the cornea is intact the eye may be bathed in the pus with impunity, but as soon as the slightest lesion appears in the epithelium infection takes place, for pneumococci are present in the pus of the lacrymal sac. In addition to this, dacryocystitis is a disease of the laboring classes, among whom ulcus serpens also prevails, while it is rarely met with in the better situated classes of society. Dacryocystitis must be considered a serious disease because of this danger with which it threatens the eye.

When speaking of simple strictures I said that the etiological treatment of diseases of the lacrymal passages was of little value. This is true to even a greater degree in dacryocystitis. The dacryocystitis usually persists, or readily recurs, whether we treat the nasal disease or not, therefore treatment must be local. Many attempts have been made to do away with the strictures by the passage of probes, and to arrest the inflammation of the mucous membrane by irrigation with such astringents as weak solutions of zinc, or such antiseptics as hydrargyrum oxycyanatum, 1 : 2000 or protargol 1 to 4%. When this treatment is to be tried the irrigation must be performed regularly at intervals of eight or ten days in connection with the passage of probes, and must be done with great care. The canula must be in a good position, so that salt solution stained with fluorescin flows smoothly through it out of the nose, before the antiseptic fluid is introduced, for if the wall of the lacrymal sac be injured, or a false passage created, this remedy will cause a severe and persistent œdema about the sac. When pneumococci were present in the pus I have tried irrigation of the lacrymal sac with a dilute solution of pneumococcus serum instead of an antiseptic, and prefer such a solution to any other, but a real and permanent cure of dacryocystitis is obtained by irrigation in only a very small percentage of cases, for the unfavorable hygienic conditions, the lack of cleanliness, and the existing nasal disease cause recurrences very easily. Moreover, the entire treatment with probes is painful, demands several weeks, and many patients avoid it. I do not understand how this form of treatment of dacryocystitis can still be called successful in modern textbooks. The facts show, on the contrary, that it is one of the most unsuccessful forms of ophthalmic therapy. In numerous cases of chronic dacryocystitis no cure can be obtained in this conservative way. There are the cases of ectasia of the lacrymal sac in which the probes may pass quite smoothly, revealing no appreciable stricture, and yet recovery cannot take place because the elasticity of the wall of the lacrymal sac has been lost by atony, and so the most important factor for the conduction of the tears is absent. Even when no such atony is present many cases of dacryocystitis can no longer be cured after the disease has lasted several years.

It is also impossible to cure a dacryocystitis when bony strictures are present, which result from syphilitic, or tuberculous, nasal disease. If we touch bare bone with the probe we need make no further attempt to preserve the lacrymal sac, and I will quote here the words that Kuhnt uses in his "Therapy of Conjunctivitis Granulosa."

"If patients who have been discharged apparently cured after a tedious and painful course of treatment with probes are seen again a year later it will be found in more than nine tenths of the cases that the supposedly cured disease is not only as bad as ever, but is worse, whenever possible."

This form of local treatment is of benefit to only a few patients. In the great majority, particularly among people of the laboring class, nothing but the operative removal of the diseased lacrymal sac should be considered. Very many eyes can be protected against ulcus serpens by the timely removal of this source of infection. It is to the credit of *Axenfeld* that he energetically advocated this prophylaxis of ulcus serpens, and most modern ophthalmologists are convinced of the prophylactic value of this operation.

The Extirpation of the Lacrymal Sac

The lacrymal sac may be extirpated under local anæsthesia after thorough disinfection of the surrounding skin. We inject 2 ccm of a 2% solution of novocain, to which a few drops of adrenalin have been added, subcutaneously in the neighborhood of the lacrymal sac. The skin is made tense by being pulled toward the outer canthus, so as to render distinct the situation of the ligamentum canthi internum. An incision is then made through the skin at the inner canthus, beginning about 0.5 cm above the ligamentum canthi internum, running downward with a gentle curve outward 2.5 cm, to a point 0.5 cm above the commencement of the bony canal, laying bare the white, firm tissue of the ligament of the internal canthus. The top of the lacrymal sac lies behind and extends somewhat above this ligament. If the lacrymal sac is not yet visible we feel for the crista lacrimalis of the lacrymal bone and make an incision along its edge, which brings into view the bluish gray anterior surface of the lacrymal sac. Next we dissect free the inner wall of the sac with a small raspatory, and then we have to decide, from the size of the sac, whether to cut through the ligament of the inner canthus or not, in order to get at the top. Many operators avoid injuring this ligament, I usually divide it. We now seize the lacrymal sac and free the apex and posterior surface with a pair of scissors curved particularly for the purpose. The most difficult part, if one may be permitted to speak of difficulties in this little operation, is the dissection of the outer surface of the sac, which is directed toward the orbit. I prefer to do this with a delicate knife rather than with scissors. After the sac has been freed on all sides it is lifted up and cut away with scissors at its entrance into the bony canal. We now use a sharp spoon to scrape the place where the sac has lain, to make assurance doubly sure, and curette out the nasolacrymal duct, not only to provide for drainage into the nose, but also to remove all traces of mucous membrane, which must not be allowed to remain, as they will interfere with the healing of the wound. The cavity is then washed out with salt solution, inspected to see that everything is right, and the wound is then closed with a few sutures. The hemorrhage, which sometimes is considerable, must be arrested. The after treatment is very simple. During the first day the cavity of the wound is firmly compressed by a dressing and bandage. The sutures are removed in from five to seven days and collodion may be applied for several days longer.

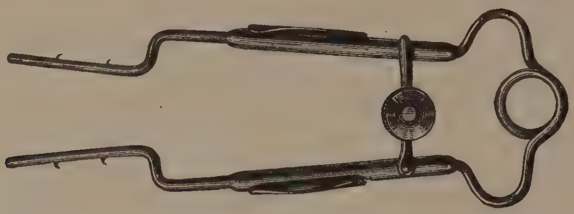

FIG. 82.—*Mueller's* Retractor for Extirpation of the Lacrymal Sac.

Like many other operators I include in this operation the extirpation of the palpebral lacrymal gland. Anatomical researches have shown that the excretory ducts of the orbital lacrymal gland are connected with the palpebral gland, and therefore it is conceivable that when we extirpate the palpebral gland the excretory ducts of the larger orbital gland are destroyed with it. In this way we may diminish the function of the larger gland and so avoid the epiphora that results from the removal of the lacrymal sac, for it is to be expected that the orbital gland itself will undergo atrophy when its excretory ducts have been destroyed. We meet with an atrophy of the lacrymal gland in extensive cicatricial contraction of the conjunctiva, which is evidently caused by the occlusion of the excretory ducts. *Schirmer* found that after the extirpation of the palpebral lacrymal gland the secretion of tears was not only diminished, but entirely abolished.

We need now to consider the behavior of the eye after extirpation of the lacrymal sac. It is self evident that the conduction of the tears is completely abolished after the removal of the sac, but this conduction has been destroyed previously. Besides, the continual inflammation of the conjunctiva and the reflex irritation of the lacrymal gland, that existed before the operation, have been done away with, so that the eyes do not lacrymate as much after the operation as was to be feared.

When such cases are studied more carefully it is found that a drop will overflow the lid margin only about once an hour while the patient is in the house, and in many cases the production of tears is still less. The patients need to wipe their eyes only at long intervals, as long as they remain indoors. It is therefore a characteristic statement of persons who have had this operation performed on them that they prefer to remain in the house. Such eyes lacrymate much more out of doors, and therefore it is advisable to remove the palpebral lacrymal gland at the same time, when the operation is to be performed on people who have to work in the open air. Even then the eye is by no means dry, for the conjunctiva produces a sufficient quantity of fluid to keep the eyeball moist. When it is also borne in mind that many dangers to the eye are obviated by this means, this little operation cannot be too strongly recommended for the treatment of chronic dacryocystitis, in spite of the slight inconvenience occasioned by the epiphora.

The cosmetic effect could not be better. A priori it would be thought that a deforming scar would remain after the extirpation, but I assure you that it is difficult to detect the cicatrix fourteen days after the operation has been performed.

This little child has a *congenital dacryocystitis.* If we press upon the lacrymal sac pus is evacuated through the puncta. but the eyes are otherwise healthy.

Ordinarily the nasolacrymal duct opens shortly before birth, but sometimes it does not. Bacteria soon settle on the eye of the infant and pass with the tears into the lacrymal sac, along with mucus and desquamated epithelium, and in a short time a decomposition of the secretion and a catarrh of the lacrymal sac begins. It is of practical advantage to know that the prognosis of this form of dacryocystitis is very good. We need only to teach the mother to press the contents of the sac out regularly and the passage into the nose will soon open spontaneously. If the suppuration should not disappear in a few days the stenosis may be easily removed by the careful passage of a probe. Spontaneous recovery is rare in the chronic dacryocystitis of adults, but it is the rule in the congenital form.

110. Dacryocystitis Phlegmonosa and Fistula of the Lacrymal Sac

This patient states that he has had epiphora of the right eye for years, and that he has long been accustomed to press pus out of his lacrymal sac. This history makes it certain that he has been suffering from a chronic dacryocystitis. Two days ago he was seized with headache, fever, and severe pain in the region of the lacrymal sac, which became swollen and red. The clinical picture now presented is not that of a simple, painless dacryocystitis, not that of an ectatic lacrymal sac; it is that of a phlegmonous inflammation.

I. Clinical Picture and Differential Diagnosis of Dacryocystitis Phlegmonosa

The skin in the vicinity of the lacrymal sac is swollen, tense, reddened, and is tender to the touch over the swelling. The inflammatory swelling extends to the lower lid, which looks œdematous. The yellow pus can be seen through the skin over the lacrymal sac. There is no doubt that this is an abscess near the point of rupturing externally.

This might be a subcutaneous abscess lying in front of the lacrymal sac, caused by infection of a wound in the skin, or a furuncle, but in that case the patient would not give a history of chronic lacrymal trouble. Such abscesses in fact do occur outside of the lacrymal sac, but in such a case the lacrymal tract must be normal and we can syringe fluid from the punctum through the lacrymal duct.

Another possibility is that it might be an empyema of the frontal sinus or ethmoid that had broken through the tissues. In regard to this I wish to call attention to one clinical point: If we look carefully at our patient we shall see that the inflammatory swelling is confined to the region below the ligamentum canthi internum and extends but slightly above it. The swelling produced by the rupture of an empyema is situated for the most part above this ligament.

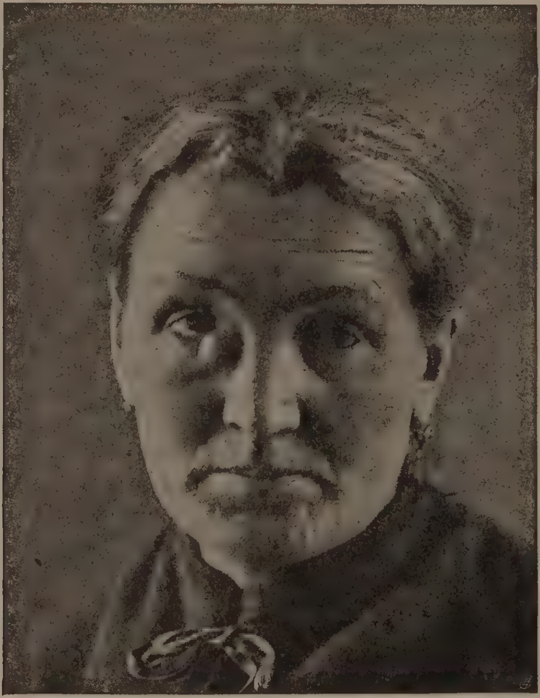

FIG. 83.—Abscess of the Lacrymal Sac.

We therefore conclude from the characteristic position of the swelling and the equally typical history that the cellulitis is etiologically connected with the chronic dacryocystitis.

II. Origin and Course of Dacryocystitis Phlegmonosa

We understand by dacryocystitis phlegmonosa a purulent inflammation of the connective tissue about the lacrymal sac. It begins in the majority of cases as a chronic catarrhal dacryocystitis. The mucous membrane of the lacrymal sac has been yielding a purulent secretion for a long time, often for years, because of a stricture in the nasolacrymal duct, and finally there comes a day when the pus agents, which have hitherto settled only in the fluid contents, and on the epithelium of the lacrymal sac, pass through the mucosa and penetrate into the surrounding connective tissue. In this form of origin, which is the most common, the abscess is only an advanced stage of a chronic catarrhal dacryocystitis. It can be readily understood that lesions may take place in the mucous membrane, as the result of the continuous suppuration, through which pus agents, especially streptococci, may find their way into the submucous connective tissue and the subcutaneous cellular tissue, and it must also be acknowledged that such an abscess frequently follows a probing of the lacrymal duct, when the probe has produced the lesion.

On the other hand I have also examined cases in which the lacrymal passages had always been healthy, and yet in which an abscess of the sac suddenly developed and perforated externally, leaving an open communication with its interior. In these rare cases the pus agents that happened to enter the sac with the tears found their way through the mucous membrane and penetrated into the surrounding cellular tissue, although there was no stricture or previous dacryocystitis.

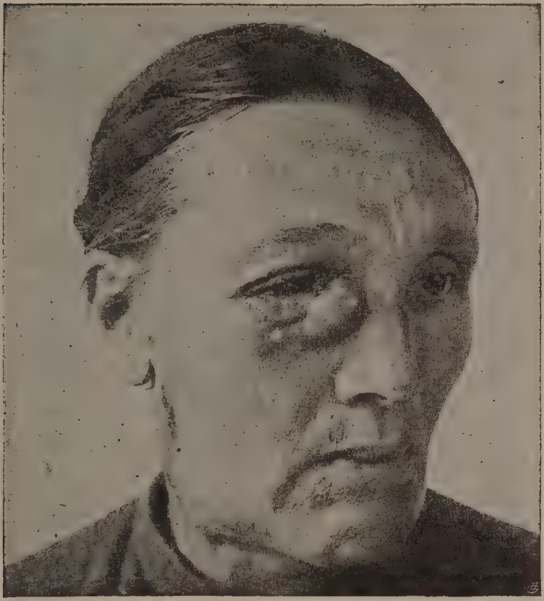

FIG. 84.—Abscess of the Lacrymal Sac. Also in *Axenfeld's* Textbook.

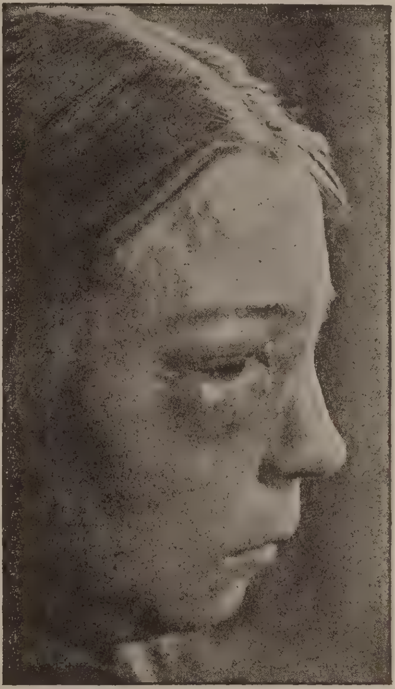

FIG. 85.—Phlegmon of the Lacrymal Sac with Extensive Swelling of the Lid.

Severe pain sets in as soon as the connective tissue about the lacrymal sac is infected, and the inflammatory swelling spreads rapidly, so that the diagnosis is unmistakable. In this stage the inflammation may still undergo involution under the influence of moist, hot compresses, but usually it goes on to the formation of an abscess. The breaking down of the tissue into pus extends, the skin becomes yellowish and finally perforation outward occurs. The perforation is usually situated in a very characteristic place below the ligament of the inner canthus, where we have to look for the anterior wall of the lacrymal sac. In many cases, however, the perforation takes place farther down toward the cheek, when the abscess had burrowed somewhat before the perforation occurred. We will now relieve the pain of our patient as quickly as possible.

III. Treatment of Abscess of the Lacrymal Sac

An attempt may be made in the earliest stages of an abscess of the lacrymal sac to bring about an involution of the inflammation by rest in bed and moist, hot applications, and this may often be accomplished, but all violent manipulation must be avoided. I do not think that the application of ice cloths, which have been recommended, is suited for this purpose. When such an inflammation is influenced beneficially it is through the hyperæmia, not by its reduction. I do not consider rational the expression of the sac and the slitting recommended even in the later textbooks. When the inflammation is not very malignant we may leave it alone during the first stage, but if it extends and forms a cellulitis the abscess is to be opened.

We render the skin somewhat anæsthetic with a spray of ethyl chloride, and make an incision at least 2 cm long, so as to open freely the abscess cavity. This incision should be made rather to the nasal side and not toward the margin of the lid, for otherwise an ectropion of the lower lid may easily be produced by the cicatrization when healing takes place. After the abscess has been opened we tampon the wound with gauze. The swelling will go down in a few days if the patient is kept in bed and moist compresses are applied; then it can soon be determined whether the wound closes after the evacuation of the pus and the lacrymal sac can be saved. Such a result is possible in many cases, but we must always consider whether it is for the best interests of the patient to save such a lacrymal sac, and this question is seldom decided in the affirmative when account is taken of the aptitude of the dacryocystitis to recur, and of the dangers that threaten the eye. If we wish to make the attempt we will pass probes after a few days and wash out the pus. At the same time the wound will be tamponed more loosely and its closure awaited. If, however, the preceding dacryocystitis had existed a long time, if the lacrymal sac is ectatic, or if insuperable strictures exist, extirpation must be performed after the inflammation has receded. The hemorrhage is greater, as a rule, than in other cases, and the entire thickened wall of the abscess must be carefully dissected away so as to save as much skin as possible. We tampon the wound for a few days and then either suture secondarily, or await healing by granulation. The result is usually good. Careful precautions must be taken that not a particle of the wall is left behind in the enucleation of such a lacrymal sac, for if the least bit of the mucous membrane remains it may continue to secrete and cause a recurrence.

Another consequence of an abscess of the lacrymal sac may be seen in this case. The essential feature of the clinical picture presented in this child is an opening below the region of the lacrymal sac surrounded by reddened skin, from which trickles a watery fluid mixed with mucus. This is a lacrymal fistula; a pathological communication between the interior of the lacrymal sac and the skin. This communication can be proved by the injection of some salt solution stained with fluorescin into the lower canaliculus, which escapes through the fistula. Sometimes the mouth of the fistula is temporarily occluded by dried secretion, so that the fluorescin does not appear, but this must not arouse the hope that the fistula is permanently closed, for the spontaneous healing of a fistula is one of the greatest of rarities. The wall of the fistula consists in great part of mucous membrane which secretes. When the trouble has lasted a long time the fistula may become so very small that only a minute opening appears in the skin, from which a drop occasionally exudes upon the cheek. It is then called a capillary fistula. The purulent catarrh of the lacrymal sac has then disappeared, nothing remains of the inflammatory redness of the skin, and in many cases it is necessary to make pressure on the sac in order to express a drop from the fistula and so locate its opening.

A fistula is produced by a perforation of an abscess of the lacrymal sac, but it must be remembered that the abscess may burrow as far down as the upper lip, or laterally beneath the skin of the cheek, before perforation takes place.

The treatment of a fistula is as follows: If it still contains large granulations it is curetted with a sharp spoon, tamponed, and the wound is allowed to become clean. Then the fistula is extirpated together with the lacrymal sac. In the case of a capillary fistula, spontaneous healing is prevented in part by the fact that it is lined with epithelium, when there is no inflammation present. If an attempt is to be made to save the sac the strictures in the nasolacrymal duct must first be removed, then the fistula may be excised and the wound closed with sutures, but such a removal of a lacrymal fistula is difficult, and it is best to associate it with extirpation of the entire sac.

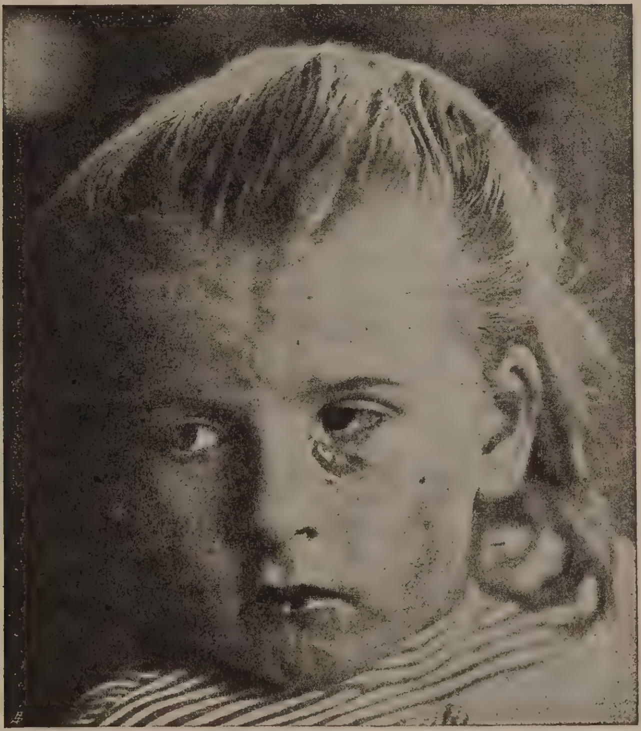

FIG. 86.—Fistula of the Lacrymal Sac.

When we mentally review the diseases of the lacrymal passages that have been described we recognize that the epiphora, the strictures of the nasolacrymal duct, the chronic catarrhal dacryocystitis, the abscess of the lacrymal sac, and the lacrymal fistula are only individual links in a chain of diseases.

This is a branch of ophthalmology in which the general practitioner can take an active part by timely recognition of the trouble, by proper care and by timely treatment.

111. Diseases of the Lacrymal Gland

Diseases of the lacrymal gland are very rare, although affections of the lacrymal sac and of the nasolacrymal duct are common. The principal reason for this is that the continuous stream of fluid from its efferent ducts prevents the entrance of microorganisms into the tissue of the gland, even though inflammatory agents are constantly present in the conjunctival sac, and, therefore, when we meet with an inflammation of the lacrymal gland we usually suppose that the infectious germs reached it endogenously. The gland is also protected from injury by its position beneath the margin of the orbit. Its diseases are divided into two groups, inflammations and tumors, which may be differentiated by the history.

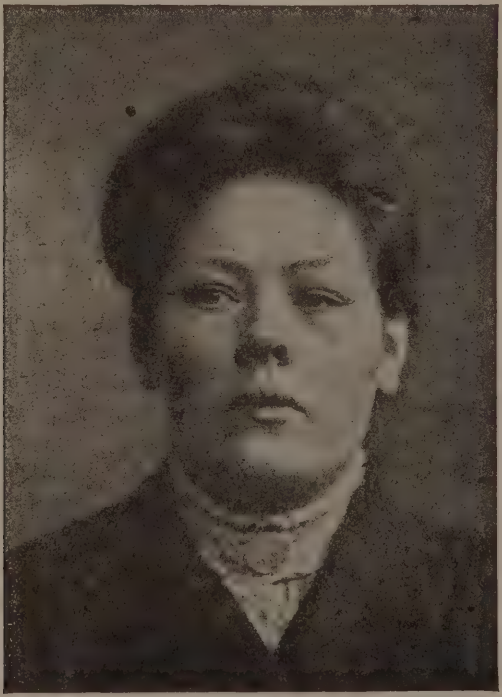

Fig. 87.—Dacryoadenitis of the Left Eye.

I. Inflammations of the Lacrymal Gland

This patient, a man of middle age, has a gonorrhœal urethritis. He has had for several days a painful swelling of both knee joints, and also an acute swelling in each upper lid, associated with some fever. The outer part of the upper lid of each eye is somewhat swollen. The palpebral fissure is rather small in its outer part and has a very characteristic form, resembling a paragraph. The outer portion of the conjunctiva bulbi is rather chemotic. The swollen, very sensitive palpebral lacrymal gland may be felt distinctly beneath the outer part of the lid. Although at first glance this may be taken to be an inflammation of the lid, yet the fact that the affection is bilateral, together with its connection with the joint disease and the characteristic localization, indicates that it is a metastatic disease of the lacrymal gland due to the gonorrhœa. We order quiet rest in bed, wet packing to the joints, and moist, hot compresses on the eyes. If the inflammation goes on to the formation of an abscess, an incision will have to be made into the gland.

Other inflammatory agents besides gonococci may excite dacryoadenitis purulenta by metastasis. More or less acute inflammations of the lacrymal glands have been observed after influenza, measles, scarlet fever, diphtheria, and typhoid fever, but these cases are rare. I wish, however, to call attention particularly to an inflammation of the lacrymal gland that is analogous to, and occurs simultaneously with, the inflammation of the parotid gland, known as parotitis epidemica, it is called mumps of the lacrymal gland. Rare cases of tuberculosis of one or both lacrymal glands have been met with; the insidious course of such a case will indicate its nature. It has been mentioned already that the lacrymal gland may become atrophic when its emunctory ducts have been occluded by cicatrization. Lacrymal fistulæ are seen in rare cases, when the pus in an abscess of the gland has ruptured externally.

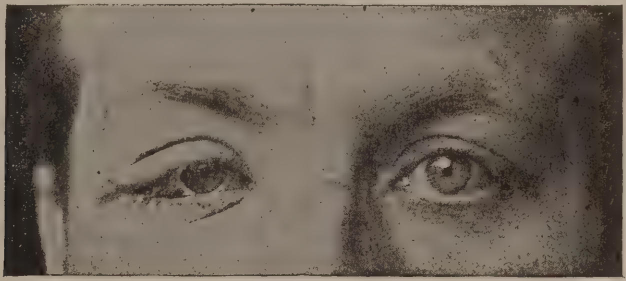

FIG. 88.—Dacryoadenitis of the Right Eye.

II. Tumors of the Lacrymal Gland

First among these come the peculiar cysts that start in its emunctory ducts. A globular, tense, painless tumor, which is enlarged by weeping, is found in the outer angle of the upper transition fold. Such a tumor must be removed by extirpation of the cyst wall. Another benign tumor is the lymphoma, which is usually bilateral, occurs in the course of a leucæmia, and is quite amenable to treatment with arsenic. Sarcoma and adenosarcoma are the principal malignant tumors met with. They occur usually only on one side. We are frequently obliged to perform *Kroenlein's* resection of the outer wall of the orbit in order to obtain convenient access to these tumors, and to remove them.

X

Diseases of the Orbit

112. Exophthalmos, the Cardinal Symptoms of Diseases of the Orbit, and the Method of Diagnosis in Such Diseases

The most prominent symptom presented by this patient is a high degree of exophthalmos, and I wish to impress indelibly two things: first, that exophthalmos is the cardinal symptom of most diseases of the orbit; second, how to discover the cause of the exophthalmos in a given case and so make the diagnosis. We shall see repeatedly that the change in the position of the eyeball is the most important and most common symptom that betrays the presence of disease in the orbit.

I. Diagnosis of Exophthalmos

It is necessary that we should know what is understood by the term exophthalmos, and be able to differentiate the condition from one in which the exophthalmos is only apparent. The enlargement of the very myopic eye may readily simulate exophthalmos; when the walls of the orbit are imperfectly developed the eye gains a certain prominence; in corpulent persons the increase of the orbital fat may cause a protrusion of the eyes, and an apparent exophthalmos may be present when the development of the two orbits is asymmetrical, but we do not need to intervene in such conditions.

We understand by exophthalmos, not a displacement of the apex of the cornea, nor a difference in the size of the eyeballs, but a displacement of the entire eyeball forward in the orbit.

We must first know the normal position of the eyeball if we are to correctly diagnose such an exophthalmos. When we look at the healthy eye of our patient we note that the apex of the cornea does not protrude beyond the margin of the orbit. Under normal conditions the eyeball does not lie exactly in, but some millimetres laterally from, the axis of the orbit, and is therefore somewhat nearer the temporal wall, while it is equally distant from the roof and the floor. It is secured in this normal position by a number of contrivances, a knowledge of which is essential before the diseases of this region can be understood.

The position of the eyeball, without regard to its size, is fixed in the first place by the cubic capacity of the orbit, which may be compared to a pyramid lying on its side, its base corresponding to the entrance of the orbit, its apex to the optic canal. In adults the cavity is about 4 cm deep and its cubic contents is about 30 ccm. Its size varies at different ages. In infants, in whom the accessory sinuses of the nose have not yet been developed, the orbit is relatively narrower than in adults. The size of its cavity, and the relations between its form and the development of the face is of great importance to the position of the eye. The ratio of its height to the breadth of its base, expressed in figures, is known as the orbital index. The studies of *Ambialet*, *Ash*, and *Fuerst* have shown that the orbit is flatter, and the eye deeper in brachycephalia, while in dolichocephalia the eyeball is generally more protuberant.

The position of the eyeball is fixed not only by the cubic capacity of the orbit but also by the mass of tissue contained in it, especially by the orbital fat and the vessels. The orbital fat serves not only as a protection for the nerves and muscles, but also as a cushion for the eyeball. In old people the fatty tissue becomes partially atrophic, so in emaciated and old persons we see the well known hollow about the margin of the orbit and the somewhat sunken eyeball. Inversely in corpulent persons the eyeball is made prominent by the increase of the orbital fat. Still, this cushion is not the most important factor that holds the eyeball in place; the retraction of the eyeball is quite moderate, even in high degrees of cachexia and atrophy of the cushion of fat. The character of the orbital vessels, especially of the veins, is of much greater importance. The orbital veins have no valves, and they are connected posteriorly with the venous sinuses of the brain, for the superior and inferior ophthalmic veins empty into the cavernous sinus after they have passed through the superior orbital fissure. Anteriorly they are united with the anterior facial vein and the external jugular, and the superior ophthalmic vein is connected with the ethmoidal vein of the nasal cavity. Thus the orbital veins have two outlets, one posteriorly into the cerebral sinuses, the other anteriorly into the facial veins. The eyeball must be forced forward whenever the orbital veins are swollen.

The ocular muscles naturally are preeminent among the means to hold the eyeball in position; the four recti endeavor to draw the eyeball back into the orbit, while the two obliques try to draw it forward. The tonicity of these two groups of muscles are normally in equilibrium. We see that this is actually so in operations for strabismus. If we divide a rectus muscle a slight degree of exophthalmos is created because now the power of the obliqui preponderates.

Finally, the fascia of the orbit is by no means the least important factor in the maintenance of the position of the eyeball. The septum tarsoorbitale is stretched like a diaphragm in front of the entrance to the orbit, and is bulged forward somewhat by the orbital fat toward the lid. The eyeball is attached to this septum by bands of fascia that come from the ocular muscles. This fascia is particularly well developed in the anterior part of the orbit; it surrounds the globe, under the name of *Tenon's* capsule, and forms a sac in which the eyeball moves as in a ball and socket joint. *Koenigstein* sums up all these things in the following words: "The eyeball is so harnessed in the orbit that in its movements it always remains in its place, i.e., it rotates about one point of rotation. This is possible only through a combined action of all factors, which are the capsule of *Tenon*, the reflected layer of the muscular aponeurosis, the subconjunctival fascia, the fat, both lids, the ocular muscles and ligaments, the vessels and the nerves."

Still, the normal position of the eyeball in the orbit is not immutably fixed; it changes its position, although but slightly, under purely physiological conditions.

Johannes Mueller ascertained that the eyeball advanced somewhat when the palpebral space was dilated, and ascribed this phenomenon to the innervation of the two obliqui, but more recent investigations by *Birch-Hirschfeld* make it probable that several factors cooperate to produce this result. When the palpebral fissure is narrow the orbicularis opposes the pressure of the contents of the orbit and presses gently on the eyeball. If this action is relaxed the eyeball may give way before the pressure of the tissues of the orbit. The active contraction of the levator also is to be added to the dilatation of the palpebral fissure, as it tends to stretch the septum orbitale. As the latter bulges forward its increased tension exerts pressure on the tissues in the orbit, which may be transmitted to the eyeball. *Birch-Hirschfeld* has found that when the palpebral fissure is dilated the globe is moved about 0.9 mm forward and at the same time about 1 mm downward.

Birch-Hirschfeld has also shown by means of photographs that the eyeball protrudes a little from the palpebral fissure when we stoop. This is due to disturbances in the circulation of the orbital veins caused by the bent position of the head. When the head is in this position most of the blood in the orbital veins flows toward the vena facialis, while, when the head is erect, it flows into the cavernous sinus. Compression of the facial veins when the head is bent immediately produces a great protrusion of the eyeball. The eyeball does not protrude from the palpebral fissure when the head is bent in consequence of gravitation, but rather because of the congestion behind it.

The eyeball can be caused to protrude one millimetre by compression of the jugular veins in the neck. Finally it may be mentioned that we know from *Donders*' investigations that the position of the eyes may change a little in deep inspiration with forced expiration, while nothing of the sort takes place in ordinary respiration. Thus we see that under certain conditions that are not morbid the eyeball can move slightly forward, and this renders easier of comprehension many pathological changes in its position.

In this patient there is no question concerning the abnormal protrusion of the eyeball, but cases are met with in which the recognition of a very slight degree of exophthalmos is difficult. A good observer can certainly recognize most pathological changes in the position of the eye without mechanical aid, but for doubtful cases, and for control examinations to determine whether the exophthalmos is better or worse, we need a more exact method of fixing the degree. Quite a number of different exophthalmometers have been invented for this purpose, among which the instrument devised by *Hering* and *Sattler* deserves mention. Very accurate measurements can be made with it, especially when modified as suggested by *Birch-Hirschfeld.*

Hertel's instrument is very suitable for practical clinical purposes and we use it a good deal. It consists of two movable frames containing mirrors placed at an angle of 45° to the parallel optic axes, and a gauge. These frames are adjusted with their spurs at the angles of the orbits and then the reflection of the apex of the cornea is thrown upon the gauge where it is read off by the observer. The handiness and simplicity of *Hertel's* exophthalmometer render it preferable to the other instruments.

After we have diagnosed an exophthalmos and determined its degree there is still one thing more for us to notice before we can proceed to the diagnosis of the orbital disease; whether the eyeball is displaced directly forward, or is dislocated at the same time laterally or vertically. I call attention now to these displacements for this reason: Every observer who meets with an exophthalmos with displacement downward will assume quite involuntarily that the trouble is in the upper part of the orbit, and when the position is divergent that the cause is on the nasal side. This is not necessarily the case, although it is frequently so. The eyeball may be rotated so as to be divergent by a tumor on the posterior temporal wall of the orbit.

II. Method of Diagnosis of a Disease of the Orbit

We know that this patient has an exophthalmos, we can express its degree in figures, and now we need to know its cause.

Exophthalmos has many causes. From a purely anatomical standpoint the eye may be pressed forward from its normal position in two different ways: the factors that hold the eyeball back may give way, or the space within the orbit may be reduced.

There is no difficulty in recognizing an exophthalmos due to a relaxation of the forces that hold the eyeball back in the orbit. In this class belong the slight degree of exophthalmos that may follow tenotomy of one of the recti, as well as the exophthalmos of oculomotor paresis, in which the tonicity of the recti is relaxed. It may also be mentioned that when the sympathetic is stimulated there appears, in addition to the dilatation of the pupil, the paleness of the side of the face and the increase of perspiration, a slight exophthalmos with dilatation of the palpebral fissure, which is often ascribed to the contraction of the musculus orbitalis. Probably the dilatation of the palpebral fissure suffices to explain this slight degree of exophthalmos. Inversely, we find in paresis of the sympathetic a contraction of the palpebral fissure and a slight enophthalmos. The sympathetic supplies the musculus orbitalis, so in paresis of the sympathetic the resistance offered by this muscle to the retracting power of the recti is abolished, and therefore the eyeball is drawn a little into the orbit. Hence the question arises in any given case whether the cause may be nervous. Such a cause has been excluded in the present case.

By far the commonest cause of exophthalmos is a reduction of the space in the orbit. This may be produced in two ways. One or more of its bony walls may be pressed in so as to encroach upon its space, or the contents of the orbit may be augmented.

The first question therefore in this case is: Is this eyeball exophthalmic because it can no longer be maintained in its normal position by its physiological strength, or is it due to a reduction of space in the orbit? The latter is the case. We cannot always differentiate whether the contraction of space causing the protrusion is due to the encroachment of the walls of the orbit, or to an increase of its contents. Both factors may unite to cause an exophthalmos in an inflammatory ectasia of one of the accessory sinuses. We need for further analysis a clinical classification of the forms of exophthalmos, and therefore I advise the following series of interrogations: Is the exophthalmos caused 1, by inflammation within or without the orbit? 2, by a tumor? 3, by a vascular abnormality in the orbit? 4, by a metabolic disease, or by poison? 5, by a cranial lesion?

The history is important in a case like this, and from it we learn that here the trouble may be of inflammatory origin.

Next we palpate. We can insert the little finger to a fair depth into the orbit, we notice whether we can feel any point of resistance, or cause pain, and in this way we can often obtain important data. We also press the eyeball gently backward, and can frequently draw conclusions from the tenderness, or the lessened mobility. Then we test the excursions of the eyeball; limitation of movement and diplopia are often concomitant, or secondary symptoms of an exophthalmos. We also test the function of the eye and examine it with the ophthalmoscope. Important conclusions can be drawn from the results of these examinations concerning the orbital disease.

In almost all of these diseases careful attention must be paid to the accessory sinuses of the nose, because many of them start from these sinuses. Therefore before forming an opinion concerning such cases we must know the rhinological condition and the findings on transillumination of the sinuses. We also need the assistance of the X rays in many cases of lesions of the margins of the orbit.

The entire organism should be submitted to examination, because we meet with exophthalmos due to general disease, as in exophthalmic goitre. Equipped with this preliminary knowledge we may successfully study the subject of diseases of the orbit.

113. Inflammatory Exophthalmos and the Importance to the Eye of Diseases of the Accessory Sinuses

Our patient, a man of middle age, suffered from an acute infectious disease some weeks ago. After his recovery he continued to have severe pains in the brow and at the root of the nose, to which he paid no attention until three days ago, when he says he had a chill and his right upper lid became swollen. Since then the swelling and inflammation have increased rapidly.

The right upper lid is quite red and swollen, particularly at the inner canthus. Pressure upon the bones in this region elicits pain. The eyeball protrudes considerably downward and outward, its movements are restricted, and the patient has diplopia. The vision is distinctly reduced and ophthalmoscopically we find a neuritis. In this case, the clinical history of which has been outlined briefly, the differentiation between exophthalmos due to a cranial lesion, to a vascular anomaly, to a tumor, to metabolic disease, and to inflammation, is not difficult. The cause is proved to be inflammatory by the history, the objective lesions, the acute swelling and inflammatory redness of the lid, and the rapid development of the exophthalmos. When the eyeball is displaced under such circumstances we speak of it as an inflammatory exophthalmos.

We must first briefly review the different types of inflammatory diseases of the orbit, and their differentiation in individual cases, for in this way only can we determine the origin of the exophthalmos and the nature of the disease. I have drawn up a purely practical chart, so arranged as to begin with the rare affections and pass on to the common causes. An inflammatory exophthalmos may be produced 1, by syphilis and tuberculosis of the orbit; 2, by the extension of a thrombosis of the cavernous sinus into the orbit; 3, by a tenonitis; 4, by an inflammation of the retrobulbar tissue; 5, by an inflammation of the wall of the orbit, or of its periosteum; 6, by a disease of the accessory sinuses of the nose.

I. Exophthalmos Caused by Syphilis and Tuberculosis of the Orbit

Syphilis is comparatively rare in the orbit; it forms about 0.02% of all eye diseases and between 1 and 2% of all syphilitic eye diseases; yet we cannot let the suggestion, that it may be the cause of any individual case of exophthalmos, pass entirely without notice, for a protrusion of the eyeball with inflammatory symptoms may be produced by a periostitis orbitæ, both in children with hereditary syphilis, and in adults who have acquired the disease. Even a gumma situated in the lid may cause exophthalmos. The physician does well to consider first whether an exophthalmos is caused by syphilis, because if it is, the treatment is settled. Periostitis orbitæ luetica may appear on the margin, or on the walls of the orbit.

When situated on the margin it is usually above, on the edge of the roof, where it produces a swelling of the lid with exophthalmos and displacement of the eyeball downward. The swelling is exquisitely sensitive and gives rise to spontaneous pains, which are characteristically worse at night. These pains may precede the swelling a long time and may simulate those of trigeminal neuralgia.

If the periostitis is situated more deeply in the orbit the roof is still specially predisposed to the attack. A tense swelling develops which simulates a tumor, and dislocates the eyeball not only forward, but outward. The similarity to a tumor is so great that the differential diagnosis must be weighed very carefully. One point of differentiation is that the wall of the orbit is very sensitive to pressure in this form of syphilitic periostitis. The syphilitic disease may also be mistaken for empyema of the accessory sinuses when it is on the nasal, or upper inner wall of the orbit.

Visual disturbances may be associated with the exophthalmos caused by syphilitic periostitis and the vision even may be endangered. Optic neuritis and contractions of the visual field are met with, which are due to the action upon the nerve of the inflammatory infiltration of the periosteum at the optic canal, or it may be to the circulatory disturbances occasioned in the vessels by the pressure of a gumma.

Paresis of the ocular muscles may also be present. I wish to call attention particularly to one symptom that is frequently met with in syphilitic periostitis of the orbit—a peculiar disturbance of the sensibility of the cornea. It is finally determined whether syphilis is the cause by the general examination of the body, by *Wassermann's* reaction, and by the results produced by antisyphilitic treatment. Such an exophthalmos, with its accompanying symptoms, retrogrades under the influence of large doses of potassium iodide with subsequent energetic inunctions. The prognosis is generally good, though visual trouble may persist, and even death has been known to result from orbital syphilis.

Tuberculosis of the Orbit

This likewise is a rare disease. Its most common form, and that most easily recognized, is the tuberculous periostitis of the margin of the orbit. It is met with chiefly in children and young people, and frequently follows a contusion. A tender place develops quite gradually on the margin, usually in its upper and outer, or its lower part, together with a swelling of the lid, and may go on to perforation and the formation of a fistula. Bare bone may be felt with the probe, a sequestrum may be cast off, and finally a deeply depressed scar may be left, which produces an ectropion. The prognosis is usually good when the fistula is curetted, the sequestrum removed and the diseased portions of bone chiselled away. It is quite exceptional for a case of orbital tuberculosis like this to produce an inflammatory exophthalmos, such as appears when the tuberculosis attacks the retrobulbar tissues.

Tuberculosis of the retrobulbar tissue is very rare. It differs from tuberculosis of the margin of the orbit in appearing in advanced age, as well as in the manner in which the infection takes place. A tuberculosis of the chorioid may perforate the sclera and spread into the orbit, or the infection may come from the lacrymal gland, as has been proved in several cases. Exceptionally also a tuberculosis of the lacrymal sac may extend to the nasal wall of the orbit. Cases have been observed by *Sattler* and *Cirincione* in which a tuberculosis of the optic nerve has given rise to an exophthalmos. Finally tubercle bacilli may reach the retrobulbar tissue by metastasis; in these cases the differentiation from a retrobulbar tumor is very difficult. Tuberculosis is excluded in this case by the rapidity with which the inflammatory exophthalmos developed.

It should be mentioned in this connection that an actinomycosis of the orbit has been observed in some very rare cases, in which the infection came from the mouth and the antrum of *Highmore.* These cases teach us that we should examine the pus carefully in every case of orbital abscess. Infection of the orbit with glanders and with anthrax has been observed very rarely. Tetanus of the orbit is of much greater importance, for it is not so very uncommon for tetanus spores to be carried deeply into the orbit by foreign bodies, chiefly splinters of wood, where the anaërobic bacteria find a favorable opportunity for development. In such cases an inflammatory exophthalmos develops with symptoms of inflammation of the orbit, especially when other pus agents are also present, and when the symptoms of tetanus appear, stiffness of the neck, convulsions, spasms of the muscles of the throat, and facial paresis, the cause of the exophthalmos is evident. We sometimes find in the eye a meiosis with rigidity of the pupil, due to spasm of the sphincter. I mention this kind of exophthalmos only in order to impress the fact that the wound should be carefully cleansed after an injury to the orbit. It is also wise to give a prophylactic injection of tetanus serum in all cases in which the wounds of the orbit are dirty, because experience has shown that it is too late to use this antitoxin after the symptoms of tetanus have appeared.

II. Exophthalmos Caused by Extension of a Thrombosis of the Cavernous Sinus into the Orbit

A comparatively rare form of exophthalmos is that in which the fundamental cause is easily recognized to be sinus thrombosis. This form is excluded at once in the present case, for the serious cerebral disturbances met with in that disease could not escape notice. We will discuss more in detail in another place the eye symptoms that accompany sinus thrombosis.

Marantic sinus thrombosis very rarely causes an exophthalmos. It is also comparatively rare for an exophthalmos to be produced by an otitic sinus thrombosis, which extends from the lower wall of the tympanic cavity to the transverse, or lateral sinus and causes exophthalmos only when the inflammation has reached the cavernous sinus and the ophthalmic veins. The œdema at the posterior margin of the mastoid is of diagnostic importance in otitic sinus thrombosis, as it is due to a thrombosis of the emissarium mastoideum. When we find an exophthalmos among the symptoms that indicate a sinus thrombosis the case is probably one of septic thrombosis of the sinus cavernosus. According to *Uhthoff*, inflammatory exophthalmos is present in 72% of such cases. Another point of diagnostic value is the fact that other purulent diseases of the brain scarcely ever cause a secondary inflammation in the orbit, so if we meet with an inflammatory exophthalmos with cerebral symptoms we can make the diagnosis of thrombosis of the cavernous sinus with a fair degree of certainty. The inflammation of the orbit may not follow a thrombosis of the sinus; the order of things may be reversed and a thrombophlebitis of the orbit may precede. Cellulitis of the orbit has also been known to cause sinus thrombosis, so we have to try to prevent this occurrence in all cases of abscess of the orbit by the adoption of suitable surgical treatment.

III. Exophthalmos Caused by Tenonitis

Tenonitis is one more rare disease that may cause exophthalmos, which may be excluded in this case by its peculiar clinical nature.

Tenonitis may be either serous or purulent. The characteristic features of the serous forms are: First, that, with more or less severe general symptoms and fever, the movements of the eye are attended with pain which is so severe that the patient prefers to turn his head rather than his eyes. This characteristic distinguishes tenonitis from other forms of inflammation of the orbit. Second, the conjunctiva presents a peculiar chemosis of a characteristic, pale yellow color. Third, the exophthalmos is not very marked. It may be added that the eye is exquisitely sensitive to the slightest touch. A rheumatic etiology can be demonstrated in most cases, so that tenonitis serosa may almost be looked upon as a metastasis of the still unknown agent of rheumatism into *Tenon's* space. The prognosis of this form is good, the inflammatory symptoms usually pass away in a few days under antirheumatic treatment. The interior of the eye is but rarely involved.

It is otherwise with purulent tenonitis. This is usually caused by influenza, but may be due to other diseases, or to injuries. I have seen a purulent tenonitis after an operation for strabismus. This form likewise develops an exophthalmos with pain and chemosis.

The pus is apt to perforate *Tenon's* capsule at the place of the insertion of one of the recti, so it is necessary in this form to open *Tenon's* capsule and to drain *Tenon's* space. The interior of the eyeball often suffers, either from a neuritis, or a purulent iridocyclitis. In such cases the infection of the interior of the eye is secondary, but this may be reversed, and a simultaneous infection of the uvea and of *Tenon's* space by metastasis is possible.

IV. Exophthalmos Caused by Orbital Cellulitis

Inflammatory exophthalmos may also be caused by the inflammation of the retrobulbar tissue called orbital cellulitis, which is a grave, acute disease that begins with serious general symptoms, fever and pain. The lids are much swollen, the conjunctiva is chemotic, the eyeball is usually displaced in the direction of the orbital axis, and its mobility is impaired. The protrusion of the eyeball needs no further explanation. The limitation of movement is due partly to the mechanical protrusion, partly to the simultaneous infiltration of the ocular muscles and the paresis of the nerves. The upper lid is swollen and hangs down from its own weight, but an injury to the levator and a true ptosis may be present. The pain also needs no explanation. It is well to know that it is dull and that it may be increased by movements of, as well as by pressure on, the eyeball, but points of tenderness, such as are met with in periostitis and sinusitis, are not present.

Orbital cellulitis varies a great deal in its course. Sometimes the swelling of the lid, the chemosis and the protrusion of the globe look threatening, and yet no suppuration takes place; in other cases pus perforates in a very short time through either the lid or the conjunctiva, producing a fistula that may either last a long time, or may heal quickly. Gangrene of the greater part of the contents of the orbit has been observed in still other cases. All of these variations in the course depend on the virulence and the nature of the morbific agents, as well as on other conditions. It is unnecessary to enumerate the agents met with in retrobulbar cellulitis; staphylococci and streptococci are the principal ones, but pyocyaneus bacilli, typhoid bacilli and pneumococci are also found.

Cellulitis of the orbit is an extremely dangerous disease, as is shown by the fact that the eye itself is often involved, and is rendered totally blind in 20% of the cases. Transient visual disturbances are met with in many other cases, and these disturbances may be considerable without any lesion that can be detected in the fundus. Inversely cases occur in which the vision is preserved in spite of the presence of a neuritis. Blindness is often associated with the picture of thrombosis and embolism of the retinal vessels. Retinal hemorrhages and optic atrophy may also be observed. The blindness may come on suddenly, or may develop gradually. As the visual disturbances are in no way due to the degree of the exophthalmos, for the vision may be perfect when the protrusion is of high degree, the question arises as to the nature of the disease of the optic nerve that is found in cellulitis of the orbit. A uniform explanation of this cannot be given, because both the ophthalmoscopic lesion and the degree and nature of the visual disturbance vary. Circulatory troubles, compression of the optic nerve and of its vessels by the swollen contents of the orbit, and toxic effects, all have to be taken into account. Thrombophlebitis of the central vein plays an important part. Even when we find signs of a so called embolism of the central artery in cellulitis of the orbit, they are not due to a real embolism, but to a thrombosis of the central artery, which results in simple optic atrophy. Occlusion of the central vessels is to be thought of primarily in a simple optic atrophy following an orbital cellulitis. A neuritis was situated in the posterior part of the optic nerve, when the vision improves after the involution of the cellulitis. When the visual disturbance occurs suddenly, the cause must be either an occlusion of the central vessels, or some other acute interference with the circulation.

Corneal complications are met with in orbital cellulitis; perforatiag ulcers and phthisis bulbi are not of uncommon occurrence.

When cellulitis of the orbit is bilateral, the inflammation may have passed from one orbit to the other by way of the cavernous sinus, or the venæ ethmoidales may have furnished the path of communication. One orbit may also become infected from an accessory sinus and then the other, through the corresponding sinus.

Aside from these complications in regard to the eye, orbital cellulitis is one of the most serious diseases with which we have to deal, for a considerable number of the patients, as many as 17%, according to *Birch-Hirschfeld*, die from meningitis, abscess of the brain, or thrombosis of the cavernous sinus. Investigations have shown that the anatomical nature of orbital cellulitis is that of a thrombophlebitis, and it can therefore be understood that it extends primarily by way of the veins toward the brain and there causes a sinus thrombosis. Cellulitis has also been observed to perforate the bony roof of the orbit and to reach the brain in this way. Finally, it seems evident to me that pus agents may enter the blood from such a cellulitis and bring about metastatically an abscess of the brain, or a meningitis. The inflammation has also been observed to spread to the temple and the region of the parotid gland, and there to cause abscesses.

Cellulitis is easily differentiated from a retrobulbar tumor by its sudden commencement and the grave inflammatory symptoms. The grave cerebral symptoms in a case of thrombosis of the cavernous sinus cannot be overlooked, and the œdema over the mastoid may also be indicative. The differentiation from tenonitis may be more difficult, but usually in the latter the exophthalmos is slight and the chemosis great, while in orbital cellulitis the exophthalmos is great and the chemosis slight. The differentiation of a periostitis of the wall of the orbit may present considerable difficulty, especially if it is associated with a cellulitis, but, as a rule, only one certain place in the wall of the orbit is affected in periostitis, and the eyeball is displaced laterally.

The etiology of an orbital cellulitis must be taken into account in order to make the diagnosis, for the presence of this disease cannot be assumed offhand, when we see an inflammatory protrusion of the eyeball, unless its cause is known. Three general causes operate to produce inflammation of the retrobulbar tissue.

Direct infection may result from injuries by foreign bodies and operations. The injury may be slight or severe; often it is caused by a foreign body that has been driven into the orbit. I once found in an orbital cellulitis, in a child, a piece of lead pencil 4 cm long.

An orbital cellulitis of metastatic origin has been observed after puerperal fever and other infectious diseases, but such cases are exceptional.

In most cases of orbital cellulitis we must primarily consider whether the inflammation has not spread to the tissues of the orbit from the neighboring parts.

Such an extension is recognized readily when the inflammation has spread from the eyeball. Thus in panophthalmitis the accompanying exophthalmos is caused by an inflammatory infiltration, or an œdema of the retrobulbar tissue. Likewise, the starting point of the inflammation is easily recognized when a purulent dacryocystitis infects the orbit. I have already demonstrated that the retrobulbar tissue can be easily reached from the lacrymal sac. The source of infection is also recognized immediately when an orbital cellulitis is due to a thrombophlebitis of the facial veins, as, for example, after a cellulitis of the lids.

The physician is less apt to remember that the origin of an orbital cellulitis is often elsewhere. The veins of the superior maxilla, of the buccal cavity, of the temporal fossa, and of the throat, are all connected with the veins of the orbit, and through them inflammations situated quite a distance away may excite an orbital cellulitis. Such a cellulitis may come from a parotitis, or a tonsillitis. All infectious diseases may cause first a sinusitis of the accessory sinuses and then secondarily an inflammation in the orbit. Usually, to be sure, a sinusitis produces first a disease of the bony wall and of the periosteum, but it may directly excite an inflammation of the cellular tissue of the orbit. The importance of diseases of the accessory sinuses with regard to the eye is indicated more and more in the clinical discussions of the day, and our interest in these diseases is still increasing.

V. Exophthalmos Caused by Inflammation of the Periosteum and Wall of the Orbit

An inflammatory exophthalmos can be produced not only by an inflammation of the retrobulbar tissue, but also by inflammation of the wall and of the periosteum of the orbit. A periostitis may be situated either at the margin, or more deeply on its wall.

(*a*) *Periostitis of the Margin of the Orbit*

The diagnosis of a periostitis of the outer margin of the orbit is easy. This girl, whom I have under treatment, had a firm, sensitive swelling on the upper, outer margin, in which fluctuation could be detected, when she entered the clinic. No exophthalmos accompanies a periostitis that is purely of the margin of the orbit, because the inflammation is situated in front of the septum orbitale. It is only when such a circumscribed periostitis develops into an abscess in the lid, as has happened in this case, that the condition becomes rather complicated, for it may be difficult to trace the abscess of the lid to the periostitis. Exophthalmos can be produced only when the inflammation extends to the more deeply lying portions of the periosteum, or when the retrobulbar tissue has become infected.

Generally speaking, the prognosis of a periostitis of the margin of the orbit is good. The focus of inflammation lies comparatively far from the brain, while the part of the bone affected lies in front of the septum orbitale, and is easily accessible. We need only to open the abscess freely and to drain it. If the inflammation is not properly treated at the right time, and the abscess is allowed to rupture spontaneously, a fistula may be left through which bare bone may be felt with a probe. This may lead to further complications in the way of changes in the position of the lid.

(*b*) *Periostitis of the Wall of the Orbit*

The condition produced by a periostitis anywhere else on the wall of the orbit is quite different. Its most prominent symptom is exophthalmos, to which a lateral displacement of the eyeball is usually added. Other symptoms are motor disturbances, diplopia, and such signs of inflammation as pain, fever, redness and swelling of the lids, perhaps nausea and vomiting.

A very important symptom is a circumscribed place of tenderness on the wall of the orbit, from which we can sometimes draw conclusions concerning the origin of the disease. Symptoms of orbital cellulitis may also be present.

A periostitis of the deeper parts of the orbital wall differs from a periostitis of the margin, not only in its diagnostic points, but above all in its prognosis. It is always a very serious disease. The sight of the eye is greatly endangered; blindness may be caused if the periostitis extends toward the apex of the orbit, to the optic canal. The dural sheath of the optic nerve is continuous with the periosteum, so the nerve may be compressed at this point and caused to atrophy, or circulatory disturbances may be produced in the nerve which result in optic neuritis, and central or peripheral scotomata. The exophthalmos naturally places the cornea in danger of xerosis.

Such a periostitis may have other serious consequences. A large, or a small piece of the bony wall may become necrotic, and be cast off so as to form a fistula. Such a grave periostitis may extend to the meninges and cause death by abscess of the brain and meningitis. A communication between the cavities of the orbit and of the skull may readily be brought about by necrosis, especially when it is situated on the upper wall, which is very thin. The mortality from this form of orbital periostitis is over 10%.

The treatment must therefore be very energetic. I have never contented myself with a small puncture, but have sought out the focus of pus after laying bare the margin of the orbit by an incision through the soft parts, and after detachment of the periosteum. In this way we are pretty certain to find any subperiosteal abscesses, as well as any retrobulbar foci of pus that may be present. Opening of the periorbital tissue must be avoided as much as possible as long as the retrobulbar tissue is intact. All this can be done much better when the margin of the orbit has been laid bare freely, as has been rightly maintained by *Birch-Hirschfeld,* than by the old method of puncture, which is still recommended in some textbooks. The temporary resection of the outer wall of the orbit, according to *Kroenlein's* method, may be indicated in periostitis of the temporal wall.

Periostitis of the wall of the orbit differs from periostitis of the margin not only in diagnosis, prognosis and treatment, but also in etiology.

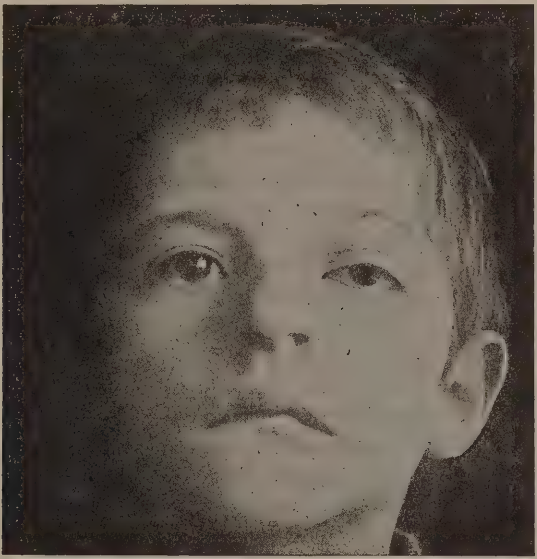

FIG. 89.—Periostitis of the Left Upper Margin of the Orbit.

Occasionally such a periostitis is met with in the course of, or as a sequel to, measles, scarlet fever, typhoid fever and angina, when the infectious agents have obtained a foothold endogenously in the bone or the periosteum. Staphylococci and streptococci are usually found in such cases of periostitis, but sometimes influenza bacilli, pneumococci and typhoid bacilli are met with. Other causes are syphilis and tuberculosis. It may also be caused by injuries; the inflammation may extend to the periosteum from an external wound of the margin of the orbit, or it may be due to a penetrating wound made by an infected foreign body. An abscess of the retrobulbar tissue may also affect the periosteum secondarily. *The most frequent cause is an inflammation of an accessory sinus of the nose.* In these cases we notice certain tender places on the wall. If the upper inner part of the orbit is sensitive to pressure, as in the present case, it is a symptom to be taken into account in the diagnosis of empyema of the frontal sinus. Hence, in the diagnosis of every case of exophthalmos due to periostitis we must seriously consider the question whether the case is really a rare one of primary periostitis, or is not rather a consequence of disease of the accessory sinuses.

VI. Exophthalmos Caused by Diseases of the Accessory Sinuses of the Nose

The most frequent of all the inflammations of the accessory sinuses of the nose to lead to disease of the orbit is that of the frontal sinus; then follow diseases of the maxillary antrum, of the ethmoid, and finally of the sphenoidal sinus. We cannot go in detail into the pathogenesis of diseases of the accessory sinuses, but I am very anxious to impress their great importance in connection with disease of the orbit. We can speak, if you wish, of a catarrh, of mucocele and of empyema, although transitional forms between these are met with, and even the acute cases are not always to be distinguished from the chronic, but it may be said that mucoceles of the accessory sinuses do not ordinarily lead to inflammation in the orbit.

Ectasiæ of the accessory sinuses occasion rather a slowly developing protrusion of the eye through the bulging forward of the bone, so this form of exophthalmos really belongs to the group which is caused by a contraction of the space in the orbit by deformation of the skull. The inner upper part of the wall of the orbit becomes ectatic most easily, in a case of mucocele of the frontal sinus, because this is the thinnest place. Sometimes such a mucocele presents considerable diagnostic difficulty, because no sign of suppuration in the sinus can be detected on examination of the nose. An ectasia of the ethmoidal labyrinth causes a dislocation of the eyeball forward and outward. These mucoceles may attain a considerable size. An ectasia of the sphenoidal sinus produces no exophthalmos. All of these things render it improbable that our case of inflammatory exophthalmos is caused by a pure mucocele.

Empyemata of the accessory sinuses, on the contrary, frequently lead to inflammation in the orbit. They may develop as acute inflammations, or they may run a chronic course from the start. When we meet with an inflammation in the orbit associated with a disease of an accessory sinus we must think primarily of an empyema of that sinus.

We need to know in what way a sinusitis is accustomed to spread to the orbit if we wish to trace back the genesis of an orbital inflammation, as in this patient in whom we have still to elucidate the fundamental disease. In a sinusitis the inflammation of the mucous membrane of the sinus interferes with the nutrition of, and extends directly to, the bone, so that in most cases in which the orbit becomes involved a periostitis, or a subperiosteal abscess, is formed first, such as I have already described. Orbital cellulitis frequently does not follow the subperiosteal abscess for a long time, but the course may be quite different, and the cellular tissue of the orbit may be involved in the inflammation very quickly. This depends mainly on the nature of the inflammatory agent. In this connection it might be mentioned that, according to *Gruenwald*, *Stanculeano* and others, the fœtid inflammations of the accessory sinuses have a buccal origin, those which are not fœtid, a nasal. The order of frequency in which the sinuses need to be taken into account is first the frontal sinus, then the maxillary antrum, then the ethmoidal labyrinth, and finally the sphenoidal sinus.

Inflammation of the Orbit from Sinusitis Frontalis

Kuhnt deserves great credit for having pointed out how important affections of the frontal sinus are to the ophthalmologist, and I advise that inflammation of the frontal sinus be thought of whenever people complain of a severe pain over the root of the nose, or the brow after catching cold. Such pains often appear periodically in a characteristic manner, and are more severe when the escape of the secretion from the sinus is rendered difficult, as when the patient is stooping, or lying in bed.

Kuhnt also showed that a circumscribed area of tenderness in its wall is of value in the diagnosis of an empyema of the frontal sinus. A dull pain is produced by pressure on the floor of the sinus from the orbit, which is distinctly different from the sensitiveness to pressure of the supraorbital nerve.

We need the aid of the rhinologist to confirm the diagnosis in uncomplicated cases. He notes the condition as to pus in the middle meatus of the nose, and may, perhaps, pass a probe into the frontal sinus, transilluminate, or take a roentgenograph. The diagnosis of an empyema of the frontal sinus in our patient has been confirmed rhinologically.

The condition presented by our patient is an advanced stage of this disease. The exophthalmos, the displacement of the eyeball downward and outward, together with the limited mobility of the eye, indicate that the empyema has broken through into the orbit. If no intervention should be made at this stage the swelling of the lid would increase, the abscess would spread along the bone and the pus would seek an exit. When this happens the perforation generally takes place in the middle of the lid, or over the ligamentum canthi internum, more rarely between the middle and outer thirds of the orbit. The perforation of the wall of the sinus varies a great deal; the opening may be very small, or a large sequestrum may be cast off. A fistula may remain when perforation has taken place through the lid.

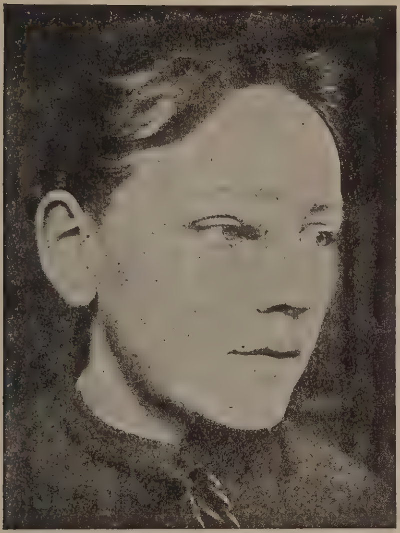

FIG. 90.—Empyema of the Right Frontal Sinus.

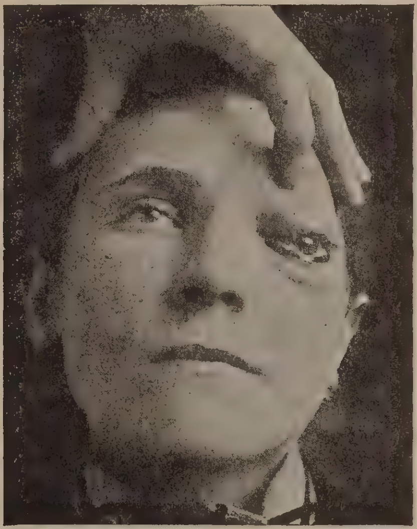

FIG. 91.—Empyema of the Left Frontal Sinus with Great Exophthalmos.

I have demonstrated this case for two reasons; particularly to impress the fact that this is a serious disease. Such an empyema of the frontal sinus may extend to the brain very readily through a perforation of the posterior wall of the sinus. The empyema may also reach the brain by way of the orbit; the periostitis attacking the roof of the latter and causing perforation at that point into the cranial cavity, with meningitis as the final result. Occasionally also a thrombosis of the orbital veins will extend to the sinus cavernosus from an orbital cellulitis.

Our patient complains of visual trouble and we find on examination that he has an optic neuritis. Choked disc, optic atrophy, and even permanent amaurosis have been observed. Diseases of the optic nerve are not so common in empyema of the frontal sinus as in diseases of the posterior sinuses of the nose and of the maxillary antrum, but they are not to be neglected. In the majority of cases the affections of the optic nerve are caused by a periostitis at the optic canal. The displacement of the eyeball occasions the diplopia. Paresis of the ocular muscles has also been observed in empyema of the frontal sinus. A number of functional eye troubles are met with, even when there has been no rupture of the frontal sinusitis, which have been ascribed by *Kuhnt* to intoxication produced by resorption of substances from the sinus. Complaints are frequent of supraorbital neuralgia, asthenopia and nervous troubles. Before we pass to the treatment let us study the differential diagnosis from inflammations of the orbit caused by diseases of the other sinuses.

Inflammation of the Orbit from Empyema of the Ethmoidal Labyrinth

The perforation of an empyema of the ethmoidal labyrinth into the orbit may take place in either an acute, or a chronic manner. In acute empyema there are serious general symptoms, the lid becomes greatly swollen, and the eyeball is dislocated outward, more rarely downward. If preparations for the perforation are made slowly a hard swelling is formed at the inner canthus, which displaces the eyeball, a crepitation like that of parchment can be felt in the region of the lamina papyracea, and tenderness in the region of the lacrymal bone may be detected by pressure on its process. This tenderness on the part of the lacrymal bone is met with more rarely than the sensitiveness of the lower wall of the frontal sinus in empyema. Orbital inflammation from ethmoiditis is excluded in this case by the site of the periostitis, but there are still other noteworthy differences between the conditions produced by ethmoiditis and frontal sinusitis.

The inflammation of the orbit takes the form of an orbital cellulitis more commonly in empyema of the ethmoid than in empyema of the frontal sinus. The vision is endangered in a greater percentage of cases for several reasons. One is that an empyema of the ethmoidal labyrinth is often associated with suppuration in the posterior accessory sinuses, which lie nearer to the optic nerve. The more common orbital cellulitis is likewise a greater source of danger to the optic nerve. A subperiosteal abscess may readily extend backward and affect the optic canal. Therefore we meet with neuritis, atrophy, and retinal hemorrhages more frequently in empyema of the ethmoid, than in that of the frontal sinus. As regards the prognosis of orbital inflammations following empyema of the ethmoidal labyrinth, it may be emphasized that a necrosis of the roof of the orbit may be produced directly by such an inflammation. The mortality attending orbital inflammation caused by empyema of the ethmoidal labyrinth has been calculated by *Birch-Hirschfeld* to be 6%.

Inflammation of the Orbit from Empyema of the Maxillary Sinus

According to *Birch-Hirschfeld*, empyema of the maxillary sinus is the cause of almost 20% of the cases of inflammation of the orbit. This form is differentiated from those due to empyema of the ethmoid, or of the frontal sinus by the fact that a subperiosteal abscess develops in the lower part of the orbit. The cheek and the lid swell and exophthalmos appears, but the eyeball is displaced upward, and there is a corresponding limitation of movement downward. Sometimes fluctuation is felt at the lower margin of the orbit, but the abscess may rupture in the upper inner margin, or at the outer canthus.

The orbital inflammation caused by empyema of the maxillary sinus runs a tempestuous course as an orbital cellulitis, in very many cases, because of a thrombophlebitis conducted through the fine venous branches from the sinus maxillaris, that empty into the ophthalmic vein. This we see also in cases in which a periostitis of the superior maxilla is produced by affections of the teeth, which may extend directly to the orbit without involvement of the antrum. There is also on the anterior surface of the superior maxilla a venous plexus which is in communication with the inferior ophthalmic vein through the vena ophthalmofacialis. A striking clinical fact is that orbital cellulitis originates more often from empyema of the maxillary sinus, the etiology of which is itself different from that of empyemata of the other sinuses. The fact is very interesting that an orbital inflammation is particularly apt to occur in those empyemata of the maxillary sinus that originate from diseases of the roots of the teeth through an inflammation of the alveolar process. Thus it is that in any given case of inflammatory exophthalmos we must think of carious teeth and of their consequences.

These inflammations of the orbit from empyemata of the maxillary sinus are also very serious diseases; they have a mortality of 15%. Visual disturbances are likewise more frequent than in orbital affections from diseases of the frontal sinus and ethmoid; neuritis, atrophy, and thrombosis of the central artery and vein are met with. With the exception of the cases in which the vessels are occluded these troubles are due to inflammation at the optic canal.

Inflammation of the Orbit Caused by Empyema of the Posterior Cells of the Ethmoid, and of the Sphenoidal Sinus

The involvement of the orbit in empyemata of the posterior cells of the ethmoid and of the sphenoidal sinus is of great interest, because intimate anatomical relations exist between these cavities and the orbit. Diseases of these posterior accessory sinuses cause exophthalmos, swelling of the lids and orbital cellulitis with comparative rarity, much more often they cause visual disturbances. A knowledge of this fact is important, because the objective and subjective symptoms of diseases of these sinuses may be very uncertain. Our attention may be called to a disease of the posterior accessory sinuses of the nose by a series of visual disturbances, for blindness, either temporary or permanent, occurs in over 40% of these cases. *We must think of an empyema of the posterior ethmoidal cells and of the sphenoidal sinus whenever we meet with a fairly rapid onset of visual trouble in one eye, for which we can discover no other plausible reason.*

The ophthalmoscopic findings vary a great deal in these cases. We may find optic neuritis, or in rare instances occlusion of the vessels, but even when the ophthalmoscopic appearance is perfectly normal a test must be made to determine whether a central scotoma for color is present, for cases are on record in which the detection of such a scotoma was the first step toward a correct diagnosis. How this central scotoma is brought about in empyema of the posterior accessory sinuses we do not know. The importance of the early recognition of such a disease is shown by the fact that empyema of the sphenoidal sinus is very apt to cause purulent sinus thrombosis, more rarely to excite meningitis and abscess of the brain.

Finally, rhinological experience has proved that empyemata may occur not only in single sinuses, but in several simultaneously, but in such cases the extension of the inflammation into the orbit takes place from one sinus. The situation of the place of the perforation and the direction of the displacement of the eyeball will permit the origin of the inflammatory exophthalmos to be recognized.

Therapy of Inflammation of the Orbit Caused by Disease of the Accessory Sinuses

Beyond doubt treatment of both the inflammation in the orbit and the sinusitis is needed, and the question is raised whether the ophthalmologist should undertake the treatment of both. It seems very natural that he should not only treat the orbital inflammation, but also attack the accessory sinus, for in the sinus is concealed the fundamental disease. Why should not the ophthalmologist undertake the treatment of the sinusitis? There are indeed some ophthalmologists who consider diseases of the sinuses to be a field for the conquest of ophthalmology, and *Kuhnt* has laid down rules for the treatment of the same, but, enticing as this surgical activity is to the ophthalmologist, who must otherwise be contented with minor operations, and little doubt as there is that some ophthalmologists are masters in this region, yet I am convinced that we must impose a certain restriction here, and I agree fully with *Birch-Hirschfeld* that the treatment of the accessory sinuses of the nose should be transferred to the rhinologist. It is desirable, and even necessary that the ophthalmologist should be conversant with the diseases of the accessory sinuses, and I believe that I have demonstrated enough of orbital surgery to make me exempt from the suspicion that I am ignorant of the technique and could not perform the operation of their evacuation. I have had sufficient opportunity to assist Professor *Hoffmann* in the operative treatment of disease of these sinuses, but I have learned that we must resist the temptation to open and evacuate the accessory sinuses from the orbit. The opinions of rhinologists concerning the necessity of radical operations are still much divided, but one thing is certain: The radical operation is not in all cases the only right way in which to cure a sinusitis. The experience of rhinologists proves that quite a number of diseases, for example of the frontal sinus, can be cured by the use of more conservative endonasal methods. The first decision to be made in any particular case of empyema of the frontal sinus is whether a radical operation is indicated. This decision can be made only by the rhinologist. Our operative ability in this region, therefore, has its limits. We are not competent to operate on empyemata of the posterior accessory sinuses, the posterior ethmoidal cells and the sphenoidal cavity. If the exophthalmos is caused by such an empyema we can only open the orbit freely, when symptoms are present that indicate imminent danger from an orbital cellulitis and the disburdening of the orbit must not be delayed. If the case is one of subperiosteal abscess due to empyema of the maxillary sinus, we may first open this up freely and drain; then we may entrust the treatment of the suppuration in the antrum to the rhinologist, for we are not competent to decide whether a conservative method of treatment, or the operative opening of the antrum is best. In our present case of orbital inflammation with an empyema of the frontal sinus we will proceed in the following manner: We will lay bare the inner and the upper inner margin of the orbit, elevate the periosteum, so as to evacuate the pus, and carefully investigate the condition of the bony wall. If the bone does not appear to be diseased the radical operation on the frontal sinus does not seem to be immediately indicated, for it has been shown that spontaneous recovery may take place in acute cases of frontal sinusitis. The sinusitis is certainly not cured by the evacuation of the orbital abscess, and the rhinologist may undertake the further treatment, after we have removed the danger immediately threatening the eye. The same rule holds good when, after opening the orbit, we find a necrosis of the bony wall, or conditions which cause a perforation of the posterior wall of the sinus to be feared. First deal with the orbital disease and then turn the patient over to the rhinologist for a radical operation. I cannot describe the details of the radical operation for empyema of the frontal sinus in this place. The methods chiefly employed are those of *Kuhnt* and of *Killian*.

Diseases of the accessory sinuses of the nose must be thought of primarily when we have to deal with an inflammation in the orbit. They are responsible for more than 60% of the cases of inflammatory exophthalmos. What a change from the theories formerly held! Never forget to examine the accessory sinuses in every case of disease in the orbit. This rule will lead to the saving of many eyes and the preservation of many lives.

114. Exophthalmos Caused by Tumors, and Kroenlein's Operation

This patient is a laborer, 35 years old, who tells us that his right eye has gradually become totally blind. His parents are healthy and he has never been seriously ill. He states that as a soldier, thirteen years ago, the sight of his right eye was not as good as that of his left, that from that time on it gradually grew worse, and that the eye has been totally blind for the past eight years. During the past two years he has noticed its gradual protrusion from the orbit, and this has finally brought him to us. There is no history of an injury.

The left eye is healthy and has perfect vision. The right eyeball is considerably dislocated forward and is at the same time deviated somewhat upward. *Hertel's* exophthalmometer shows that the apex of the right cornea is 13 mm farther forward than that of the left; the eye could easily be luxated from the orbit. It is totally blind, not divergent, freely movable in all directions and accompanies the excursions of the other eye. An attempt to press the eyeball back into the orbit succeeds only to a slight degree and is not painful. The eyeball itself is of normal size. The margin of the orbit is normal. A soft, circumscribed, almost fluctuating mass can be felt, by palpation below the eyeball, 1 to 1½ cm behind the lower margin of the orbit; it has no connection that can be detected with its floor. The patient has difficulty in closing his lids over the eye, which is moderately irritated. With the ophthalmoscope we find a genuine atrophy of the papilla with sharp outlines. The patient must have a retrobulbar tumor.

The principal symptom of a retrobulbar tumor is an exophthalmos. The farther back in the apex of the orbit the tumor is situated the later is the appearance of this principal symptom. Tumors which develop in the funnel formed by the muscles, or in the apex of the orbit, drive the eyeball forward in the direction of the optic axis, while neoplasms that start on a lateral wall, or near the anterior segment of the orbit, displace the eyeball laterally; but we cannot diagnose with certainty the situation of the growth from the lateral dislocation, because the eyeball may have been rotated.

Limitation of mobility may form a second symptom. This may be due partly to mechanical causes, such as the lack of space, and partly to affections of the muscles and nerves.

The third symptom consists of the changes in the optic nerve. The signs of engorgement in the papilla may be produced by compression of the nerve, and we find the result of neuritis in the form of atrophy as well as, occasionally, of retinal hemorrhages. The eyeball may be depressed by the growing tumor; myopia may be caused by an elongation, hypermetropia by a shortening of its axis. Other symptoms that may be associated, such as a swelling of the lids and a dilatation of the orbital veins, depend on the situation, the nature and the extent of the tumor.

I need not picture in detail the results produced by a particularly malignant tumor. As the neoplasm continues to grow the cornea may be destroyed by keratitis e lagophthalmo, in consequence of its imperfect covering by the lids. The eyeball may be completely surrounded by the exuberant growth. In other cases the mass of tumor may press out of the palpebral fissure, bleed, become gangrenous and putrefactive, or penetrate into the accessory sinuses, and cause death by proliferation into the cranial cavity, or by metastasis. Plain as is the clinical picture of an advanced orbital tumor, the diagnosis may be very difficult at the commencement of the disease. Frequently we are obliged to palpate the orbit under general anæsthesia and to transilluminate the sinuses.

Syphilis in the orbit may simulate a tumor, so it is a clinical maxim, which should never be forgotten, to always examine for syphilis in a case of exophthalmos in which a tumor is suspected. A point of value in the differential diagnosis is that syphilitic neoplasms in the orbit usually cause severe pains, which are worse at night, while tumors, as a rule, cause no such pains. Furthermore, the orbital wall is generally sensitive to pressure in gummatous periostitis, while this symptom is ordinarily absent with tumors. Another point of weight is whether other luetic symptoms can be demonstrated, especially concretions on the bones of the face and skull. Above all *Wassermann's* reaction must be determined in every case of orbital tumor, as this tells us with certainty whether we have to deal with a syphilitic patient or not. Finally it can do no harm to institute an energetic antisyphilitic treatment at once in a case in which the diagnosis is uncertain.

It is not such a very rare occurrence to meet with a case in which the presence of an orbital tumor is indicated by all the symptoms, and yet no tumor is found on operation. These diseases are still obscure. In part they are due to chronic inflammation in the orbit. *Birch-Hirschfeld* has studied such inflammatory lymphomatoses of the orbit, with the formation of follicles and infiltration of the orbital tissue, which may run courses with symptoms that markedly resemble those of an orbital tumor.

Finally, before we operate, we must remember that exophthalmos occurs in general diseases. Tumors, known as lymphomata, appear in the orbit in leucæmia and pseudoleucæmia. These need not be operated on, because experience has taught us that they will undergo involution spontaneously under the influence of quinine and arsenic. All other tumors must be operated on whether they are benign or malignant, but the methods of operation vary. We try to reach small tumors situated anteriorly through the conjunctiva, or through an incision in the lid. When the tumor is advanced and malignant the orbit must be exenterated. The question always arises whether the eye can be saved, either because it is capable of vision, or for cosmetic reasons.

We have still to consider what tumors we are likely to meet with in the orbit, without much reference to whether they are benign or malignant, because in many cases that differentiation can be made only at the operation. We may divide orbital tumors into several groups for purely practical diagnostic reasons: 1, vascular growths; 2, cysts; 3, bony tumors; 4, tumors of the soft tissues of the orbit; 5, tumors of the optic nerve.

Various kinds of *vascular growths* occur in the orbit. They may be telangiectases, which start in the lids and extend deeply into the orbit, or cavernomas, which are situated most frequently in the muscle funnel. I mention these vascular growths first because in many cases their diagnosis is easy. We see the convolutions of vessels through the skin, the tumors usually grow slowly, and the exophthalmos is often increased by stooping and shouting, conditions that increase the engorgement in the vessels. Encapsulated angiomata are also met with, in which this change in the fullness of the vessels does not take place.

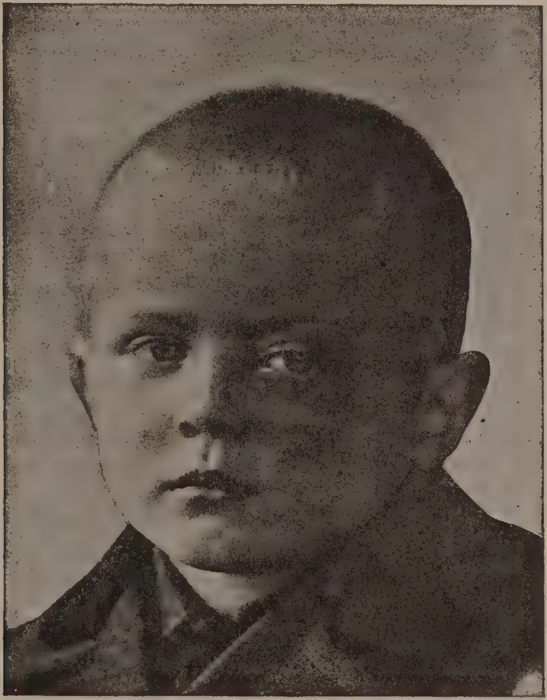

FIG. 92.—Pseudotumor of the Left Orbit: Eye Very Protuberant and Dislocated Downward; *Kroenlein's* Operation; No Tumor Found.

Lymphangiomata also occur in the orbit; the cavernous angioma is usually congenital.

Cysts of the orbit are far more common. This is particularly true of dermoid cysts, which are formations due to fœtal invagination of the outer germinal layer. They are usually situated outside of the muscle funnel and exhibit a certain predilection for the vicinity of the anterior opening of the orbit, especially in its upper and outer part. The growth of these tumors varies, they differ considerably in size, and some have been seen as large as a goose egg. As a rule they contain epithelium, cholesterin, hair, bone, or a fluid oil. Other orbital cysts with serous contents are met with, as well as blood cysts, which originate from encapsuled hemorrhages. A cyst may also originate from the bursa mucosa of the trochlea.

Echinococci have been removed from the orbit in rare cases. Such a cyst consists of a mother cyst with secondary vesicles, which has been caused by an embryo that has wandered into the cellular tissue of the orbit. It is characteristic of these tumors that they are accustomed to excite pain and inflammation in the surrounding tissue during their development. A positive diagnosis before operation can be made only by an examination of the fluid of the cyst, which is characterized by a high specific gravity and the presence of hydrochloric and succinic acids. The cysts develop usually from the sheaths of the muscles and of the optic nerve.

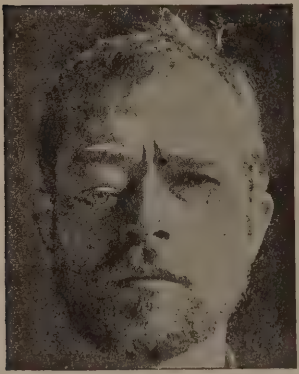

FIG. 93.—Osteoma of the Posterior Wall of the Frontal Sinus.

A cyst may also be formed above the inner canthus, which is known as an encephalocele, because its wall is composed of dura mater and its contents of cerebrospinal fluid. This congenital cyst usually protrudes through the suture between the frontal bone, the nasal process of the superior maxillary, the ethmoid, and the lacrymal bones, but it may occur through a dilated foramen opticum, or in the roof of the orbit through the superior orbital fissure. A pulsating exophthalmos may be simulated when it is situated in the roof of the orbit. Pulsation is generally to be noticed in these hernias of the dura mater. Such a cyst may also be recognized from the fact that we can press out part of its contents, which passes back into the cranial cavity and produces the symptoms of increased brain pressure. Encephaloceles are also found that are closed and separated from the cranial cavity. Portions of the nasal mucous membrane may also be constricted off toward the orbit during fœtal life, and cysts may form from their remains at the inner canthus, the walls of which are lined with laminated cylindrical epithelium and goblet cells. They contain mucus.

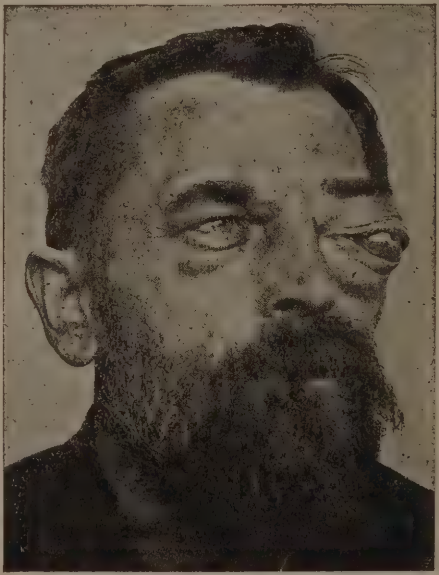

FIG. 94.—Fibrosarcoma of the Orbit.

Other congenital cysts of the orbit that lie behind the upper lid are generally met with in connection with microphthalmos. When the mesodermal tissue, that takes part in the closure of the fœtal optic cleft, proliferates, cysts are sometimes formed which may be connected with the interior of the eyeball by a slender cord, or may be entirely separate. Traces of the retinal elements are to be found within them.

A plexiform neuroma is met with in extremely rare cases. According to Panas these tumors come chiefly from the branches of the lacrymal nerve, and the twisted cords extend from the lids to the orbit.

When the question as to the presence of any of these rare or congenital forms is settled, we investigate whether in any given case the tumor comes from the bones of the orbit. Two forms of bony growths are met with in practice; exostoses and osteomata of the accessory sinuses.

Exostoses, called ivory exostoses because of their hardness, are generally situated on the roof, or the upper inner wall of the orbit. They may develop from embryological rudiments, or as the result of injuries.

Osteomata usually start in the accessory sinuses and penetrate into the orbit. The osteoma of the frontal sinus is the most common. When such a tumor starts from the sphenoidal cavity it makes its presence known first by alterations in the optic nerve. Sometimes it happens that the tumor becomes separated from its matrix and lies freely movable in the sinus. When a tumor like this develops in a sinus it presses the wall of the orbit before it in the same manner as a mucocele. Osteomata originate from the periosteum, or from remains of fœtal cartilage. When in the frontal sinus they are apt to be bilateral. The prognosis of bony tumors is good.

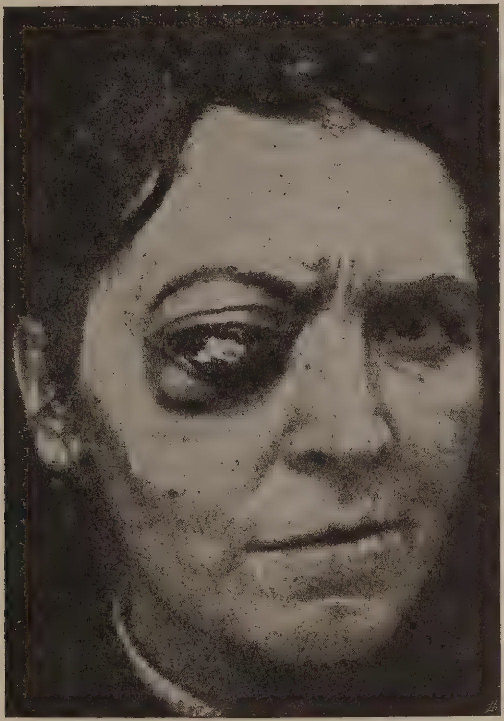

FIG. 95.—Melanosarcoma of the Orbit with Metastases in the Skin.

If we find no indication that the tumor comes from the bony wall we have to consider whether it is one of the soft parts of the orbit, and it is of importance in the differential diagnosis to know that *carcinoma of the orbit* originates either from the lids, in very rare instances from the conjunctiva, from the lacrymal gland, or from the accessory sinuses, the maxillary antrum in particular. Metastatic carcinoma also occurs, but on the whole carcinoma of the orbit is one of the rarer tumors with which we have to deal. *Sarcoma of the orbit* is much more common in practice. It is not pigmented in the great majority of cases and is composed of round or spindle cells; but other forms, such as the fibrosarcoma, myxosarcoma and endothelioma, have been observed many times. It is of clinical importance to know that the small celled sarcoma is the most malignant form and grows rapidly. The sarcoma may start from every possible tissue in the orbit, from the periosteum, from the connective tissue in the orbit, from *Tenon's* capsule, from the sheaths of the optic and other nerves, inside or outside of the muscle funnel, beneath the roof of the orbit, or close behind the eyeball. It may appear at any age.

Finally we have to consider whether or not the exophthalmos is caused by a tumor of the optic nerve. A *tumor of the optic nerve* may originate in three different ways: it may develop primarily within the optic nerve sheath, when we call it a primary tumor, or the optic nerve may be involved in a neoplasm coming from the surrounding soft parts in the orbit, from the base of the skull, or from the dural sheath, when we have a secondary tumor; or it may be caused metastatically. Metastatic carcinoma and sarcoma have been found in the optic nerve, but they are extremely rare. Although the size and consistence of primary tumors vary, yet in general they are pyriform in shape. They may involve only a portion, or the whole, of the nerve. We may clinically assume a tumor of the optic nerve to be present when exophthalmos appears early, increases slowly and without pain, and is either directly forward, or somewhat downward and outward. The eye becomes blind early, with symptoms of either neuritis or atrophy. *A. v. Graefe* called attention to the fact that the mobility of the eye is usually well preserved. A final symptom of this form of tumor is that its course is comparatively benign.

Kroenlein's Operation

Formerly we were obliged to try to remove retrobulbar growths by way of the conjunctiva, according to the method devised by *Knapp*, and a tenotomy often had to be performed on one of the muscles in order to obtain sufficient access to the depths of the orbit. A thread was then placed about the posterior part of the optic nerve by means of an aneurysm needle, the nerve was divided as closely as possible to the optic foramen, and then the attempt was made to remove the tumor from the orbit. As may be imagined, the view of the conditions within the orbit was not very good during this procedure, and the radical extirpation of retrobulbar growths was difficult. *Kroenlein* therefore rendered mankind good service when he essentially facilitated interventions in the orbit by the introduction of his operation, which he performed for the first time in 1886. The number of fortunate cases, in which retrobulbar tumors have been removed in this manner with preservation of the eye, is continually increasing.

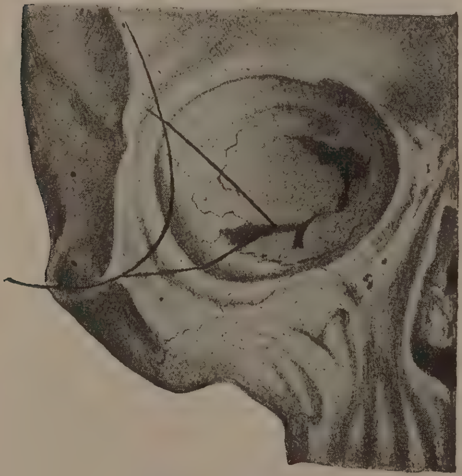

FIG. 96.—Incisions in *Kroenlein's* Operation after *Haab*. The Curved Line Marks the Site of the Incision through the Skin.

After shaving the eyebrow and the hair about the temple a curved incision is made from the place where the linea semicircularis of the frontal bone intersects a horizontal line 1 cm above and parallel to the supraorbital margin, along the temporal margin of the orbit, with its convexity toward the latter, to the upper level of the malar bone, thence backward to end in the middle of the malar bone. The middle portion of this incision, opposite the outer canthus, is carried down to the bone, while above and below only the skin, fascia and muscular tissue are cut through. The incision is about 7 cm long and lies somewhat to the nasal side of the temporal margin of the orbit, so that the latter is well freed from its soft parts. We next retract the margins of the wound and separate the periorbita from the entire outer wall of the orbit with an elevator, above to the zygomatic frontal suture, below to the inferior orbital fissure, and downward to behind the zygomatic sphenoidal suture. Now we must mark the point to which the incisions to be made in the bone will converge, by means of which we may temporarily remove the piece of bone. The best point for this purpose is the inferior orbital fissure. The soft parts are drawn aside with a strong retractor and then we cut through the periosteum over the distinctly visible sutura zygomaticofrontalis and chisel through the zygomatic process of the frontal bone. The position of this incision must be accurate, for if made too high the cranial cavity may be opened. Then the incision is carried with the chisel obliquely through the wall of the orbit, so that it is completed about 1 cm behind the anterior end of the inferior orbital fissure. While this is being done the contents of the orbit must be carefully retracted toward the nasal side. The frontal process of the malar bone is next divided horizontally with the chisel close to its base and the incision carried through the bone to the inferior orbital fissure. In this manner we have rendered movable a piece of bone, the base of which is formed by the outer margin of the orbit, while its apex lies behind the anterior end of the inferior orbital fissure, and nothing more is necessary, except to bend this

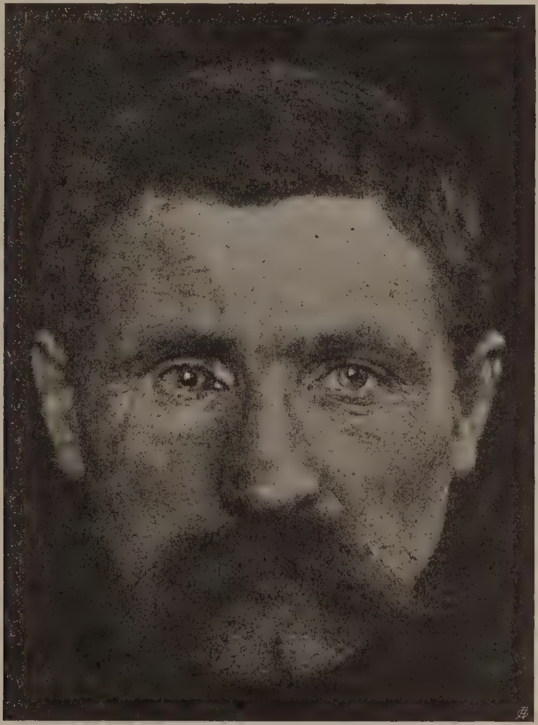

FIG. 97.—Right Eye: Preservation of Eyeball after *Kroenlein's* Operation. Operated on by *Roemer*, Tumor Described by *Loehlein.*

piece of bone outward, together with the flap of skin, fascia and muscle, and a way of access to the interior of the orbit is opened. Now comes the third and most important step. We divide the periorbita, which hitherto has covered the retrobulbar portion, and reach the tumor. The tumor in this patient is as large as a walnut, fairly well defined, starts anteriorly from the optic nerve and extends very deeply into the pyramid of the orbit, as I can tell by feeling of it with my finger. The diagnosis, that this was a tumor of the optic nerve, was therefore correct. I shall try to protect the muscles as well as I can, but the removal of the tumor and the preservation of the eyeball are the considerations of chief importance.

After the tumor has been removed we have to suture the muscles that have been divided, to replace the bone flap in its original position, and to suture carefully the periosteum and the soft parts. The lower part of the wound is drained for a few days with gauze. The dressing must be applied with great care, with attention to see that the eye is perfectly covered by the lids. If the cornea is anæsthetic a temporary suturing of the lids together is recommended by *Axenfeld.*

Our patient returns today after the lapse of two years (see Fig. 97). The result is very satisfactory. There is no sign of a recurrence, although it is still possible that this may take place. His general condition is good. There is a slight enophthalmos at the site of the operation and the movements of the upper lid are somewhat impaired. A slight upward motion is all there is left of the movements of the eyeball. This patient is very fortunate that he still retains his eye.

115. Exophthalmos Caused by Vascular Anomalies

This patient, 60 years old, goes from one clinic to another in order to exhibit the interesting lesion in his eye. In 1898 he fell a distance of three hundred metres in the mountains, striking one place after another, and sustained a grave fracture of the base of the skull, from which he made a fair recovery. Some time later, according to his statement, a swelling developed in his orbit which produced an exophthalmos that pulsated synchronously with the heart beat. Such a condition is known as a pulsating exophthalmos. He has been operated on several times in Leipsic. First, the orbital veins were ligated, later, ligation of the common carotid was performed; both operations were effective for only a short time. The eyeball has finally become fixed in the inner upper angle of the orbit, and has become changed from a pulsating exophthalmos to a pulsating enophthalmos. The eye is blind from atrophy of the optic nerve. It lies deeply in the orbit, fixed to the wall by a cicatricial cord, but is still driven forward from ½ to 1 mm synchronously with the pulse. This case illustrates the results produced by a pulsating exophthalmos, so we will briefly review the salient features of the disease.

(*a*) *Pulsating Exophthalmos*

It will be recalled that one of the different groups into which exophthalmos was divided was distinguished as caused by vascular anomalies, and the pulsating movement of the eyeball indicates that we have to deal here with one of this class of cases. The principal symptom is the exophthalmos, caused by a rupture of the internal carotid artery into the cavernous sinus. To understand the condition it is necessary to remember the anatomy of the parts involved. The internal carotid is embedded in the cavernous sinus near the clivus and the superior ophthalmic vein opens into the sinus.

Such a rupture may be produced in two ways. It may take place while the patient is stooping, or coughing, when the wall of the carotid is not normal, particularly in pregnant women. It is characteristic of this idiopathic form of pulsating exophthalmos that the clinical symptoms develop very quickly, that the patients notice a cracking sound and feel severe pain. The other form results from injuries, especially from fracture of the base of the skull, when a fragment of bone may be driven into the carotid. It is also met with after gunshot wounds, and even after severe contusions of the head.

In cases of fracture of the base of the skull the symptoms of the serious injury predominate at first, bleeding from the mouth and nose, unconsciousness, and vomiting, and it is only gradually, frequently after the lapse of weeks, or even months, that the symptoms of a pulsating exophthalmos appear. When fully developed the clinical picture cannot be mistaken. The upper lid is red and œdematous, its skin is full of dusky, dilated veins, it hangs tensely down over the exophthalmic eyeball, and the conjunctiva is chemotic. The degree of exophthalmos varies. The eyeball may be displaced directly forward, but it is usually dislocated laterally, or downward. The movements of the eye are restricted, especially outward. The dilated veins on the surface of the eye are very distinct. The cornea may be anæsthetic, but it usually remains normal as long as it is covered sufficiently by the lids.

We can press the eyeball partially back into the orbit, when a rough thrill and pulsation will be felt; a distinct blowing murmur can also be heard through the lid with a stethoscope. These symptoms vanish when the common carotid is compressed.

The patients are extremely annoyed, not only by the exophthalmos, but also by the murmur, which they hear constantly. The vision may be normal, but in many cases the circulation in the papilla is affected, either by a venous engorgement with swelling of the disc and retinal hemorrhages, or compression of the central vessels, which renders the retina ischæmic and results in atrophy, as in this patient.

Fortunately the lesion is usually on only one side, but cases have been reported in which pulsating exophthalmos was present on both sides, or in which the second eye became affected after improvement of the first.

Although the diagnosis of a pulsating exophthalmos is easy, it must be remembered that it may be counterfeited by an encephalocele of the roof of the orbit, or by a lymphangioma.

Spontaneous involution has been observed in a few rare cases. A thrombosis of the superior orbital vein extends into the sinus and occludes the laceration in the carotid. Death results in other cases from hemorrhages through the orbit and nose, but the prognosis is generally more favorable. Improvement, and even cure, can be obtained in many cases by compression of the common carotid, accomplished by means of certain instruments, but ligation of the carotid is surer, as has been proved by *Sattler*, *Nieden* and others. The pulsation and murmur disappear. Complications may certainly be produced; embolism of the central artery has been observed, and *Schmidt-Rimpler* saw a pulsating enophthalmos after the operation. Other procedures have been tried. *Golowin* tied the ophthalmic vein, after performing *Kroenlein's* operation, and *Sattler* extirpated the aneurysmal venous sac after double ligation.

(b) *Intermittent Exophthalmos*

Vascular abnormalities may cause exophthalmos in another way. Very rare cases have been reported in which the eyeball protruded from the orbit every time a venous engorgement took place within it. Such a venous engorgement may follow bending forward of the head, compression of the jugular veins, or forced expiration with the nasal aperture occluded. We call this an intermittent exophthalmos.

It is characteristic of this condition that it always affects only one eye. If a patient with this trouble stoops an exophthalmos appears, and then the eye returns to its normal position, or at least sinks back, when the head is held erect. In many of these cases the exophthalmos is produced by the turning of the head to one side so as to cause the sternocleidomastoid to press upon the jugular vein.

All of the factors mentioned indicate the presence of a varicose dilatation of the veins of the orbit, and such a varix is almost universally admitted to be the cause, but we have as yet no satisfactory explanation how these varices are formed. Many investigators, like *Causé*, and *Elschnig*, ascribe it to an inflammation of the wall of the veins. According to *Birch-Hirschfeld* it is also possible that the primary cause of the disease is to be found in an abnormal narrowness of the anterior channels for the outflow of the blood from the orbit. When the head is held upright the blood flows from the orbit into the cavernous sinus, when the head is bowed it seeks to escape chiefly through the facial veins, but if a jugular vein is insufficiently developed, or if the outflow channels are abnormally small, the orbital veins gradually become dilated by the engorgements that recur frequently for years, when they are predisposed to do so. This is at all events an explanation why intermittent exophthalmos usually appears when the patient is stooping.

If such a patient is directed to stoop, the exophthalmos will be distinct in a few minutes. The degree varies considerably. Usually the eye is driven forward in the direction of the axis of the orbit, but it may be displaced at the same time downward or laterally. This depends on which vein of the orbit is affected by the varix. If it happens to be the superior orbital vein the eyeball will be displaced somewhat downward. Sometimes it is stated that the exophthalmos appeared suddenly after severe bodily exertion, whooping cough, or a traumatism; in other cases no such cause is alleged and the condition developed gradually.

Diplopia and impairment of the mobility of the eyeball are absent in most cases in spite of the great protrusion. The central vision may suffer somewhat while the eye is protruded, but usually it becomes normal again when the head is held erect. Paleness and obscuration of the papilla have been observed while a patient was stooping, and the pupil may be dilated as long as the protrusion continues, but serious lesions in the fundus are not apt to be met with.

It is evident from the nature of its origin that the chances of recovery in intermittent exophthalmos are slight, but, on the other hand, the prognosis quoad vitam is good. No death from this cause has yet been recorded. Two complications may occur; orbital hemorrhages have frequently been observed, as might be expected with ectasiæ of the orbital veins, and the vision may finally be affected. The visual troubles are to be ascribed partly to these hemorrhages, and optic atrophy may be found with the ophthalmoscope. A rare complication is the development of pulsation of the eye in its altered position. It has been supposed by *Mulder* that, in such cases, the opening of the superior orbital vein into the cavernous sinus was dilated, in addition to the varicose degeneration of the orbital veins, so that the blood could flow back from the sinus into the vein and so communicate a pulsating movement to the eye.

It is also evident from the origin of intermittent exophthalmos that we can do nothing for such patients except to advise them to avoid everything that tends to cause engorgement of the venous blood in the orbit, like stooping, straining at stool, lifting, and compression of the neck. Improvement has been reported as the result of a careful regulation of life. No improvement in the varix in the orbit is to be expected from a pressure bandage.

116. Exophthalmic Goitre and Exophthalmos from Poisoning

This is a case of *Basedow's* disease[1] which has been sent to us because a shortening of the palpebral space is necessary in at least one eye to prevent keratitis e lagophthalmo. I cannot deal here with the entire pathogenesis of this extremely interesting disease of metabolism, it is not within my province, but will simply point out the importance of the ocular symptoms.

A marked case of this disease is characterized by four cardinal symptoms: 1, tachycardia, with pulsation of the large arteries of the neck; 2, swelling of the thyreoid gland; 3, a peculiar tremor; 4, bilateral exophthalmos.

I. Cardinal Symptoms of Exophthalmic Goitre

The tachycardia and the increase of the force of the cardiac impulse may be directly seen, but surely distinctly felt in the epigastrium.

The patient states that at times he has a feeling of palpitation of the heart, a sensation as of impending death, and a pain in the epigastrium. The pulse is not only rapid, but has a certain arhythmia, which is very marked only at times. The heart is somewhat enlarged. When we place a finger on the large arteries of the neck we feel a distinct thrill, and we hear a loud murmur on auscultation. In many cases the head suffers a rhythmic concussion, caused by the great intensity of the cardiac impulse. This symptom, which is called *Musset's*, is absent in this case.

The second symptom of the disease is a swelling of the thyreoid gland. Frequently one lobe, the right as a rule, is particularly enlarged. A distinct thrill may be felt over the gland and a roaring noise can be heard in it, with the stethoscope, which is loudest at the places of entrance of the large arteries.

The third general symptom is a peculiar tremor, the diagnostic importance of which has been recognized particularly by *Charcot* and his pupil *Pierre Marie*. When our patient stretches out his hands with the palms down, the rapid, delicate tremor is immediately seen. This type of tremor is also observed in neurasthenia and the traumatic neuroses. It is present in more than 90% of the cases of exophthalmic goitre, may be present in the lower limbs, and may even spread over the entire body. When the patient closes his eyes gently a delicate tremor of the lids can also be seen. This symptom is known as *Rosenbach's* phenomenon. It is not characteristic of this disease, but is met with both in neurasthenics and in healthy persons. True nystagmus occurs very rarely in exophthalmic goitre.

[[1] Better known in this country as exophthalmic goitre, in England as *Graves'* disease. —*F.*]

II. Ocular Symptoms in Exophthalmic Goitre

The degree of exophthalmos is usually the same in both eyes, but one eye may be more protuberant than the other, and cases have been recorded in which the exophthalmos was present on only one side. The exophthalmos has also been observed to retrogress on the side of the operation, after thyreoidectomy, but not on the other side. Such experience led *F. Mueller* to the theory that an influence is exerted by an enlarged lobe of the thyreoid upon the eye of the same side, but this is not a reliable symptom, for the thyreoid may be enlarged on one side and a monolateral exophthalmos be present on the other. The fact that the degree of exophthalmos may vary considerably throughout its course is of general interest concerning the nature of the disease. We can press the eyeball slightly backward, but as soon as the pressure of the hand is removed it returns to its exposed position. If we direct the patient to stoop, or if we compress the jugular vein, the exophthalmos seems to increase a little. *Sattler* and *Birch-Hirschfeld* have demonstrated by means of photographs that when the head is held upright compression of the facial veins exerts a distinct influence upon the prominence of the eyeball in exophthalmic goitre.

An abnormal fullness of the veins of the orbit plays a considerable part in the etiology of the exophthalmos in this disease. *Snellen* discovered by auscultation a blowing murmur over the eye, which he took to be vascular, but *Hering* has proved it to be a sound produced by the activity of the orbicularis, which can be demonstrated in every healthy person. It is most marked when the lids are tightly closed and disappears when the eye is opened slowly; it is therefore of no significance in this disease.

The eyes are protruded in the direction of the orbital axes and, as a rule, their motility is not impaired as in other forms of exophthalmos, but sometimes the patients complain of a feeling of tension and pressure. The exophthalmos, which is usually bilateral, is a very important symptom in exophthalmic goitre, especially as it frequently necessitates an operative intervention, as in this case.

With regard to the diagnostic importance of the exophthalmos it must be borne in mind that it is usually the last cardinal symptom to appear, and that it is absent in more than 20% of the cases. As to prognosis it has long been known that the degree of the exophthalmos is not directly proportional to the severity of the disease; a high degree of exophthalmos may be present with slight general symptoms, and vice versa.

When a man with normal eyes looks upward, under normal conditions, the upper lid covers a portion of the upper segment of the cornea, but the palpebral fissure is so wide in this patient that when he looks up a narrow band of sclera is still visible above the upper margin of the cornea. This is the second ocular symptom of the disease, and is that which gives to patients with exophthalmic goitre their peculiar appearance, by means of which they may often be recognized at quite a distance. It might be thought at first that this gaping of the palpebral fissure was due to the exophthalmos, but it has been proved to be wholly independent, for cases are met with in whom the palpebral fissure may gape even more widely than in this case, although no exophthalmos has as yet developed. The upper lid is abnormally retracted and this widening of the palpebral fissure may in turn increase the exophthalmos, for, as has been mentioned already, the influence of the widening of the palpebral fissure in the production of a slight protrusion of the eyeball has been recognized since the researches of *Joh. Mueller*.

It is a matter of clinical importance that the retraction affects only the upper lid, and that the voluntary closure of the lids is usually possible in spite of it; the palpebral fissure is closed imperfectly during sleep only when the exophthalmos is very great.

Another lid phenomenon that may be demonstrated on this patient is *v. Graefe's* symptom, by which we understand a disturbance, or an abolition, of the associated movement of the upper lid with the depression of the visual plane. I direct the patient to follow quite naturally with his eyes the movement of my hand as I slowly move it downward from a horizontal, or better a slightly elevated, visual plane. Under normal conditions the upper lid descends in proportion with the movement of the eye, but in our patient the upper lid descends very little, or not at all. When the eye is depressed still further the upper lid makes jerking movements to accompany the eye, but it never reaches its normal level. If I now move my hand upward the eyes alone rise at first, but after the visual plane has reached a certain level the upper lid follows quite promptly. This symptom is therefore to be perceived only when the eyes are made to move downward.

If we try to evert the upper lid while the eyes are looking downward we will find that this is not accomplished as easily as on the normal eye. *Gifford* believes that this is a noteworthy symptom of exophthalmic goitre, but it is due to the fact that the upper lid remains behind when the eye looks downward and is somewhat retracted, conditions which render eversion of the lid difficult. Frequently there is also a swelling of the skin and of the subcutaneous cellular tissue. I question the diagnostic importance of this peculiar lid symptom.

As a rule the two lid symptoms are not absent when exophthalmos is present, but they may be demonstrable in cases in which the protrusion of the eyeball has not yet developed. They have also been observed in one eye alone when there was no protrusion. When the exophthalmos is monolateral the lid symptoms are accustomed to be present only on the same side, but they may not be marked, or may be altogether absent, even when the exophthalmos is of high degree. They are demonstrable in about two thirds of the cases.

To estimate correctly the diagnostic importance of the lid symptoms it must be borne in mind that there are persons in whom the upper lids retract, so that the palpebral fissure may gape a little, during intent fixation and lively conversation. A faulty associated movement of the upper lid in looking downward has been met with in healthy people by many observers, but this symptom is very rare in healthy persons, so it is of diagnostic importance.

The physician must also take into account that *v. Graefe's* symptom in particular has been observed in other diseases of the nervous system, and that it is also met with in paresis of the ocular muscles. In oculomotor paresis of one eye that is improving, the upper lid may readily fail to accompany the movements of the eye downward, especially if the affected eye executes a movement of convergence simultaneously with its depression. This phenomenon has been termed the pseudo-*Graefe* symptom, on account of its similarity to the lid symptom met with in exophthalmic goitre.

Furthermore, the palpebral fissure may gape abnormally when, in a bilateral associated paresis of the superior recti, these muscles react somewhat to innervation. The increased impulse necessary in looking upward is transmitted to the associated elevation of the lid and acting on the levators, which are not paretic, naturally results in an increased upward movement of the lid.

Finally, the palpebral fissure may be made to gape in a monolateral paresis of the superior rectus associated with the blepharoptosis. The abnormally strong innervation of the weakened superior rectus induces, by the associated innervation of the levator, a greater elevation of the lid, while this abnormal innervation acts upon the other eye, in consequence of the secondary diversion to it. I have seen such a case of paresis of the superior rectus and inferior oblique, in which the difference in the widths of the two palpebral fissures was very distinct when the visual plane was lowered, but all these complications are comparatively very rare and do not detract from the diagnostic value of the lid symptoms in exophthalmic goitre.

A fourth eye symptom is the extremely infrequent reflex closure of the lids. While we normally wink from five to ten times a minute, albeit at unequal intervals, a patient with exophthalmic goitre winks scarcely once a minute. This is called *v. Stellwag's* symptom. It is not so frequently pronounced as the other lid symptoms.

A number of attempts have been made to explain the occurrence of these three lid symptoms, the gaping of the palpebral fissure, the disturbance of the associated movement of the lid as the visual plane is lowered, and the derangement of the reflex closure of the lids in winking. There have been held answerable for them 1, an increased contraction of the smooth muscles of the lid; 2, a diminished tension of the orbicularis; 3, a lesion of a hypothetical centre of co-ordination for the levator and orbicularis on the one hand, of the superior and inferior recti on the other; 4, mechanical factors due to the anatomical construction; 5, an increased tonicity of the levator palpebræ superioris. The last theory, which was advanced by *Moebius*, is the most probable. If we suppose the tonicity in the levator to be increased we have a natural explanation of the tendency to dilatation of the palpebral fissure, we can also understand the inhibition of the downward movement of the lid, and that this tendency toward dilatation of the palpebral fissure has an inhibitive effect on the reflex winking of the lids.

These are the important eye symptoms, which are of diagnostic value in cases of exophthalmic goitre, but quite a number of others are connected with the disease, which I will briefly review.

Little lateral displacements of the light streak, and fluctuations of calibre of the retinal vessels, synchronous with the radial pulse, can be seen with the ophthalmoscope in very rare cases. They were observed first by *Becker*. The phenomenon has no diagnostic value by itself, for it is rare in *Basedow's* disease, and occurs in insufficiency of the aortic valves, as well as in chlorosis and anæmia. Just such anæmic conditions are frequently met with in this disease. Otherwise no characteristic symptoms are to be found within the eye. The statements that the disease may cause not only a prominence, but also an enlargement of the eye and myopia are not defensible. The behavior of the pupils, of the accommodation and of the visual field are absolutely normal. The vision is not affected, with the exception of one interesting phenomenon; optic atrophy and neuritis have been observed in some cases, and *Coppez* has shown that visual disturbances which present the clinical picture of tobacco amblyopia may arise after the prolonged and excessive administration of preparations of the thyreoid. *Birch-Hirschfeld*, as well as *Nobuo-Inouye*, has been able to produce in dogs an atrophy of the optic nerve with pronounced symptoms of degeneration in the retina by long overfeeding with thyreoidin. Although we cannot consider here the entire treatment of exophthalmic goitre we may advise care in the use of this remedy. There is little of interest within the eye of our patient, but he has some external eye symptoms.

These patients frequently complain of lacrymation for which no external, local cause can be found. Severe attacks come on even during the night, although normally the secretion of tears ceases during sleep. This must have a nervous origin. The opposite phenomenon also is not rare; the patients complain of a feeling of dryness in the eye and the eye is less moist than it should be. This may be partially explained in cases with great exophthalmos by the fact that more fluid evaporates from the larger surface of the eyeball which is exposed, but patients who have no exophthalmos likewise complain of this symptom and we must therefore suppose that the secretion of lacrymal fluid is diminished. Whether this is due to a lessened working power on the part of the lacrymal gland remains to be proved. Finally we will come back to the symptom on account of which the patient was sent to the clinic.

The imperfect covering of the cornea, in consequence of the retraction of the upper lid and the exophthalmos, combined with the infrequency of winking, has produced a lesion on its surface, the further development of which must be prevented. The surface of the cornea appears rather dull and lustreless. Superficial infiltrations may develop in such cases and lead to ulceration and perforation. We must deal with such a keratitis in a way similar to that employed in keratitis neuroparalytica. The drying of the upper layer of epithelium prepared the way for secondary infections. The eye should be protected by an ointment during the night in order to prevent this injury. In this eye we shall have to abbreviate the palpebral fissure.

Still more severe forms of keratitis are met with in this disease, but fortunately they are rare, and they cannot be ascribed wholly to the drying of the cornea. Both corneæ may break down into pus within a very short time. Such a very grave keratitis has been observed in cases not attended by exophthalmos, in which dryness could not be the exciting cause. I think *Sattler* is correct in the theory that toxic substances probably circulate in the blood of such patients, and that they impair the nutrition of the corneal tissue and lower its power of resistance to bacterial infections in a way similar to that which I pointed out in the discussion of keratomalacia. An increased pressure upon the cornea must therefore be avoided in grave cases of this disease; hence the field of application of shortening the palpebral fissure has its limits.

There are only two more ocular symptoms to be taken into account in connection with this disease. *Moebius* pointed out the presence of an insufficiency of convergence, which has accordingly received the name of *Moebius'* symptom. According to *Sattler* such an insufficiency of convergence is comparatively rare in *Graves'* disease, and the conditions of dependence of the insufficiency upon the refractive condition of the eyes must be considered. When a patient with this disease has a high degree of myopia, with which an insufficiency of convergence innervation is associated, it cannot be spoken of as a specific symptom of exophthalmic goitre, but a latent insufficiency may readily be made manifest by nervous exhaustion.

Finally, it must be mentioned that pareses of the ocular muscles may be met with in grave cases of this disease, especially in the form of bilateral ophthalmoplegia externa. Pareses of associated muscles have also been observed. Pareses of individual muscles occur, but they are rather rare.

I will say, in connection with this case, that there are other general diseases, and cases of poisoning, in which we meet with exophthalmos as a symptom. One of these diseases is acromegaly. According to *Hertel* this symptom is due to an abundant proliferation of the orbital fat. *Birch-Hirschfeld* saw acromegaly start with an attack of exophthalmos, and he believes that the tumor of the hypophysis caused circulatory disturbances in the orbit by compression of the cavernous sinus.

Cases have been observed in which exophthalmos developed after the prolonged use of preparations of thyreoidin, and exophthalmos can be produced experimentally in this manner in rabbits and monkeys, as has been proved by *Cunningham* and *Edmunds*. *Birch-Hirschfeld* did not succeed in obtaining the same result in dogs.

There is however a poison, paraphenylendiamin, with which a high degree of exophthalmos can be produced regularly in dogs. If a dog is injected subcutaneously with 0.06 gram of paraphenylendiamin hydrochlorate to each kilogram of body weight, a great exophthalmos will develop within a few hours. The drug also causes in the animals an increased secretion of tears and mucus, together with a considerable elevation of the intraocular tension. The exophthalmos is produced by a great œdema of the orbital cellular tissue, as shown by the anatomical researches of *Grunert*. The poison causes an acute engorgement of lymph in the orbit. It is well to be acquainted with this effect of paraphenylendiamin, because it is used as a cosmetic for dyeing the hair.

I have instilled some drops of a strong solution of cocain in one eye of this patient to conclude the demonstration. When the two eyes are compared a dilatation of the palpebral fissure of the cocainized eye can be perceived, and the eye gives the impression that it is driven forward somewhat, and that it is staring. *Lewin* and *Guillery* believed this exophthalmos to be produced by the relaxation of the reflex tension of the orbicularis and by stimulations of the terminal filaments of the sympathetic in *Mueller's* muscle, but *Birch-Hirschfeld* has shown that this exophthalmos is no greater than the protrusion of the globe in passive dilatation of the palpebral fissure. Hence we have to consider the dilatation of the palpebral fissure to be the only cause of the exophthalmos produced by cocain. This effect of cocain decides the question whether the staring of an eye depends mainly on the dilatation of the palpebral fissure. As soon as the palpebral fissure gapes widely we consider the eye to be staring; it has been displaced forward only a little.

117. Oxycephalia and Exophthalmos

This child has a congenital deformity of the skull, a typical oxycephalia. Such cases are of interest for two reasons: optic neuritis and atrophy are caused in a large percentage of the cases, although the optic nerves are normal in this patient, and bilateral exophthalmos is often present. It is necessary to remember the intimate relations that exist between the eye and the orbit in order to understand the reason for the exophthalmos in oxycephalia. When the eye of a child is enucleated the orbit remains backward in its development; inversely a deformity of the orbit influences the position of the eyeball. We see this exemplified in cases in which a high degree of exophthalmos results from ectasiæ of the walls of the accessory sinuses.

The configuration of the orbit is changed in oxycephalia; it is considerably shortened by a frontal position of the greater wing of the sphenoid. The origin of the deformity was ascribed by *Virchow* to a premature ossification of the sutura coronaria, and *v. Michel* was the first to describe a case of oxycephalia with exophthalmos. In most cases the exophthalmos has only a diagnostic interest and is slight, but occasionally it may be so great as to give rise to a keratitis e lagophthalmo.

Uhthoff has called attention to the fact that exophthalmos may occasionally occur in hydrocephalus, but this rarely happens, because the abnormal intracranial pressure acts uniformly in all directions and presses the sutures apart instead of pressing in the roof of the orbit. Still it has been shown in cases with exophthalmos that such a depression of the roof of the orbit may be produced by both hydrocephalus internus and externus. I may finally mention that in extremely rare cases an exophthalmos has been observed in connection with rhachitic lesions of the skull, produced by the depression of the roof of the orbit by the simultaneously existing hydrocephalus.

XI

Glaucoma

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118. Acute Glaucoma and Its Prodromal Stage

If every general practitioner would learn to recognize immediately the peculiar nature of the disease from which this patient is suffering he would accomplish much good and prevent much harm. The clinical picture is so characteristic that the diagnosis can be made without reference to the history.

About the cornea is a marked injection of a peculiar, dusky color, in the formation of which both systems of vessels are involved. Vessels from the episcleral plexus anastomose and surround the cornea like a wreath, while the vessels of the scleral conjunctiva are twisted, tortuous and surcharged with blood. The anterior ciliary veins, which accompany the recti, are abnormally distended. It is not without reason that I call attention to the peculiar color of this injection. This is no such hyperæmia as we have been accustomed to meet with in other acute inflammations; it has predominantly a venous character, the character of engorgement.

The cornea exhibits a peculiar dullness; it looks as if it had been breathed upon and has lost its lustre. Its surface looks as if it had been pricked with needles, and little unevennesses may be seen with *Zeiss'* loupe, which are really due to desquamations of the epithelium. It is also insensitive. If I touch the cornea with a twisted bit of cotton the patient does not feel it. This insensitiveness is not always uniform, but some portions may have less feeling than others.

The anterior chamber is very shallow; it is evident that the lens and iris have drawn near to the cornea.

The pupil is dilated and, in this case, it is oval. The iris is narrower above than below, and the entire pupil seems to have been displaced upward. From within it comes a gray, or greenish gray reflex.

If this does not suffice for the diagnosis, the intraocular tension must be tested. Allow me to explain how to make this test, for I have repeatedly seen it done in an erroneous manner.

I direct the patient to close his eyes gently, as if in sleep; then I place the forefinger of one hand on the inner half of the upper lid, the forefinger of the other hand on the outer half, and then test the fluctuation by pressing alternately with one finger and then with the other upon the eyeball. It is improper to direct the patient to look down during this examination, because, aside from the fact that in response to this direction patients sometimes excite spasmodic contractions of the muscles of the lid and face, which may cause quite an increase in the tension of the eye, it is possible that the turning of the eye downward may cause an increase of tension in consequence of the compressing action of the external muscles, and thus we introduce a source of error into our examination that may easily be avoided.

Numerous instruments have been devised for the purpose of giving us the intraocular tension in figures, but none of them can supplant this simple method. It has been calculated that the normal tension of the eye is equal to the pressure of 24 mm of mercury, but we have little to do with this clinically.

Every student must learn the feeling of the normal tension by practice in the examination of many patients. The tension of the human eye varies in fairly wide limits under normal conditions, and yet the physician soon learns to detect from the feeling whether it is increased or not. It is very judicious to compare the tension with that of one's own eye in doubtful cases. An accurate tonometric examination of each case should be made in the clinic.

In order to have comparative designations for the different degrees of hardness in our descriptions, we follow the suggestion of *Bowman*. We designate normal tension as Tn (tension normal) and express the increase by means of the figures 1, 2 and 3, preceded by the sign +. T + 1 means that there is a slight, but distinctly recognizable, increase of tension; when this is questionable we write T + 1(?). T + 2 means that there is a considerable increase of tension; T + 3 that the eye is stony hard, that no impression can be made on it with the finger. We find T + 3 in our patient, which completes the diagnosis.

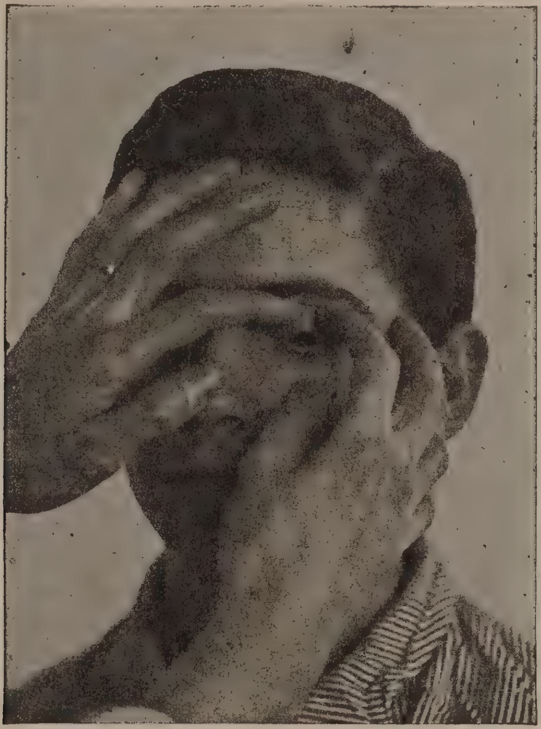

FIG. 98.—Testing the Intraocular Tension.

This condition of the eye, in which there is a considerable increase of tension, we call glaucoma. The next question is concerning the source of this increase of tension.

The diseases of the eye in which the tension is increased are divided into two large groups: those of *primary*, and those of *secondary glaucoma*. By primary glaucoma is understood an increase of tension that has taken place in the eye without having been induced by any other morbid process. Primary, genuine glaucoma is therefore a disease sui generis.

In secondary glaucoma the increase of tension is occasioned by some other affection of the eye. We have already met with it repeatedly, for instance as a result of seclusio pupillæ, and in the course of a sarcoma of the chorioid.

The first thing to be determined in this case, therefore, is whether the attack is one of primary, or of secondary, glaucoma. The history furnishes data; the patient has hitherto had healthy eyes, but the objective examination gives the final answer: we find no additional pathologic changes which can be held responsible for the great increase of tension. This increases the probability that we are dealing with a primary glaucoma, but the differential diagnosis is not yet complete.

Primary glaucoma, regarded from the clinical standpoint, appears in three different forms: 1, acute inflammatory glaucoma; 2, chronic inflammatory glaucoma; 3, glaucoma simplex. Three fundamental symptoms are common to all: 1, the intraocular tension is more or less distinctly increased; 2, the characteristic glaucomatous excavation of the head of the optic nerve develops when the disease has lasted for some time; 3, the vision fails, and blindness finally supervenes.

We need the aid of the history again to help us to differentiate these different forms. The patient says that the pains began quite acutely; they are situated both in the eye itself and in the brow and temple, radiate into the teeth of the upper jaw, extend frequently over the entire half of the head, and are often intolerably severe. The appearance of the eye by focal illumination shows it to be severely inflamed. There can no longer be any doubt that we have to deal with an acute attack of glaucoma.

When we try to look into the eye with the ophthalmoscope we find the refractive media cloudy, especially the cornea, as may be seen by focal illumination.

The assertion has been made that the lens is saturated with serum during an attack of acute glaucoma, but I believe this to be untrue and impossible. The involvement of the vitreous in the cloudiness is still a matter of debate. However this may be, an ophthalmoscopic examination can be made very imperfectly, if at all, during an acute attack of glaucoma, on account of the opacity of the refractive media.

If this were a case of chronic inflammatory glaucoma we could find and examine the optic nerve much more easily, and in glaucoma simplex the diagnosis is apt to be made first by means of the ophthalmoscope. Thus the history and the objective examination together inform us with certainty that this is an acute attack of glaucoma.

The severe pain is not the only symptom that has led this patient to consult us; he is alarmed concerning his vision. He can count fingers only a very short distance from his eye. Vision usually sinks very rapidly in an acute attack of glaucoma. The patient may still be able to read with difficulty at first, and then the vision may fall within a few hours to quantitative perception of light. When it is possible, in less severe cases than this one, to test the visual field it will be found to be contracted.

A number of cases have been reported in which the eyes were permanently blinded by the first attack of glaucoma in the course of a few days, or even of a few hours. This worst form of acute glaucoma was named by *v. Graefe* glaucoma fulminans. In the majority of cases the course and result of an acute attack of glaucoma is not so sad as this. The pains and inflammatory symptoms gradually retrogress after several hours, or days, sometimes after weeks, the cornea becomes clear and the vision improves, but the vision never becomes as good again as it was before the attack.

An acute attack of glaucoma does not pass off without leaving traces in the affected eye; certain ominous signs permanently remain from which we can recognize a primary glaucoma, or a predisposition of the eye to this disease.

The anterior ciliary veins about the cornea remain overfilled, the anterior chamber is shallower than it should be, the pupils have a suspicious tendency to dilate, and react more slowly than in a healthy eye. We find in the iris signs of commencing tissue atrophy, it is narrow and slate gray.

If an examination is made with the ophthalmoscope after the acute attack has passed away, the head of the optic nerve is seen to be much reddened. This is only a sign of the great general hyperæmia which existed during the increase of the tension.

Attacks of acute glaucoma recur after intervals of rest. They are not usually as intense as the first one, but each causes a further loss of vision, until finally the eye is rendered blind. During this time the glaucomatous excavation of the head of the optic nerve has gradually developed.

A second possible course is that the acute attack does not quite disappear. The pains and severe symptoms become ameliorated to a certain degree, but the tension remains permanently above the normal; in other words, chronic inflammatory glaucoma supervenes upon the acute attack.

No proof is needed that the promptest help is necessary in an acute attack of glaucoma. In 1856 *Albrecht v. Graefe* made the great discovery that an eye thus seriously endangered could be saved by iridectomy.

The essential symptoms of glaucoma is the increase of tension, and, if we are to understand the clinical picture, we must try to trace back to it the other symptoms.

The extremely numerous sensory nerves in the ciliary body and iris are compressed when the intraocular tension is suddenly increased to such a degree; this explains the pains felt by the patients. Heavy pressure is brought to bear on the blood vessels, especially the veins, which have the least resisting pressure within them. The principal veins that conduct most of the blood from the interior of the eye, are the venæ vorticosæ, which are easily compressed because of their oblique course through the sclera. The blood coming from the uvea must then seek another outlet, which it finds in the anastomoses of the anterior ciliary veins, and when these anterior ciliary vessels are dilated and surcharged with blood they form the thick, vascular wreath about the cornea. Thus the increase of tension explains the circumcorneal injection.

In an acute attack of glaucoma this increase of tension sets in suddenly and with great vehemence, and creates a disturbance of the circulation associated with œdematous engorgement. This inflammatory œdema causes the opacity of the cornea. It may be seen immediately after an iridectomy that this opacity is not due to an infiltration, or to a cellular exudate, for when the tension is lowered it disappears as quickly as it came.

The insensitiveness of the cornea is also easily explained through the increase of tension. The ciliary nerves are compressed and paralyzed, while the inflammatory œdema interferes with their nutrition. It has been demonstrated anatomically by *Fuchs* that the œdema spreads along the nerve fibres in the cornea, and may extend through *Bowman's* membrane even into the epithelium.

The dilatation of the pupil is partly explained by the pressure paralysis of the fibres of the oculomotor nerve. The iris and lens are pushed forward simultaneously, in consequence of the increase in volume of the vitreous, and render the anterior chamber shallow. At the same time the insertion of the iris is pushed farther forward by the displaced ciliary processes, a circumstance which contributes to the dilatation of the pupil. If this displacement of the insertion of the iris is particularly marked in any one direction, the pupil must necessarily assume an oval form.

The gray green reflex is explained by this dilatation of the pupil. We would not linger over this were it not that from it glaucoma received its name γλαυκός (sea green). This gray or greenish color of the pupil is nothing else than the normal reflex of the old lens. We see it in all elderly people when their pupils have been dilated with atropin. It is seen so distinctly in glaucoma only because the pupil is dilated so that the sclerosed, old lens reflects light more strongly.

The inflammatory hyperæmia of the head of the optic nerve and the pulsation, which is still to be elucidated, are also consequences of the increase of tension, and so is the glaucomatous excavation of the papilla, which appears later, as well as the visual disturbances, in which the cloudiness of the cornea takes an essential part.

The pressure bears most heavily on the retina. When an arterial pulse is present it shows that the cardiac systole alone is able to force fresh blood into the vessels, and when this is absent there must be a greater or less degree of retinal ischæmia as the result of the increased tension. The pressure within the arteries becomes less the farther they are from the heart, and therefore the terminal expansions of the retinal arteries are usually compressed; this results in a contraction of the visual field, which often starts in the nasal half, in a way characteristic of glaucoma. As the optic nerve and its principal artery are situated to the nasal side of the posterior pole of the eye the vessels that supply the temporal half of the retina must extend a longer distance than the others, their compression will result in a more noticeable disturbance, and the interference with nutrition in the temporal half of the retina produces a corresponding defect in the nasal portion of the visual field. Aside from the ischæmia, the retinal elements themselves suffer from the increase of tension; at least, the recent researches of *Schreiber* have made it probable that grave lesions may be produced by the hyperæmia and serous saturation of the retina. A comparatively slight pressure on the retina can cause a local reduction of its sensitiveness, as may be proved by pressing the outer wall of one's own eye with the finger, when a corresponding darkening of the visual field may readily be perceived. Taking all of these things together we see that the most essential ocular symptoms in acute glaucoma can be obtained from the increase of tension. This renders the clinical picture of acute glaucoma rather more understandable, although the entire nature of the disease has by no means been satisfactorily explained as yet.

The changes which are to be seen in the eye of this patient usually require some time for their development. During this period coming events cast their shadows before, and it is well when the physician knows these harbingers of evil, for the earlier glaucoma is recognized the more can the patient be helped.

The symptoms in the prodromal stage are both subjective and objective. As the disease usually attacks persons who are advanced in years it is not surprising that many patients come to the physician because of troubles which seem to be purely presbyopic, yet it occasionally happens that the ophthalmologist finds this presbyopia to have appeared too early. This premature presbyopia has been ascribed both to a diminution of the range of accommodation and to a decrease in the refraction, and it may be supposed that the glaucomatous process is in part the cause. In harmony with other ophthalmologists I consider it not proved that the refraction may diminish in consequence of glaucoma, for we find the condition of the refraction remaining the same even when the glaucoma has lasted for years. It has been demonstrated experimentally by *Koster* that the anterior segment of the eyeball undergoes no change of form when the tension is greatly increased, and particularly that the radius of curvature of the cornea is not increased, as it would be expected to be if the hypermetropia were increased during the glaucomatous increase of tension. Direct measurements of the human glaucomatous eye have also failed to detect any change in the radius of the cornea.

Statements have been made that astigmatism against the rule is very common in glaucoma; some claim it to be the consequence, others the cause of the disease, but no substantial reasons for either view have been advanced.

The opposite condition, an actual increase of the refraction has occasionally been seen, or supposed to exist. These claims of fluctuating refraction are of interest to the ophthalmologist alone, while the frequent clinical observation of an unusually rapid recession of the near point is of interest as well to the general practitioner, for cases have been frequently met with in which this rapid recession of the near point was noticed a long time before other signs of glaucoma became manifest. In regard to this question of premature presbyopia, we must note that hypermetropic eyes, in which the first signs of presbyopia naturally appear rather earlier, are the more frequently attacked by glaucoma; the percentage as given by statistics varies from 50 to 75.

When a very rapid onset of presbyopia is the first precursor of glaucoma, we cannot ascribe the symptom to a partial paralysis of the branch of the oculomotor nerve that supplies the ciliary muscle, because the tension is not increased. I also consider as very questionable *Schmidt-Rimpler's* idea that an increased tension of the vitreous produces a greater relaxation of the zonule. It seems to me more plausible to suppose that the nutrition of the lens is impaired in such a way as to cause a loss of its elasticity, for there is no doubt that the conditions of nutrition and metabolism in eyes that finally suffer from glaucoma have been abnormal for a long time prior to the attack. For practical purposes of diagnosis it is sufficient to know that the possibility of an approaching glaucoma should be taken into account in elderly people who complain of a rapid increase of presbyopia.

Other observations that many glaucomatous patients make for a long time before they consult a physician are worthy of attention and are of considerable importance. Our patient tells us that for a considerable length of time he has occasionally seen variegated circles about lights when he looked at them. These rainbow colors are rather characteristic, in that the outer ring is usually violet, with concentric rings of red, yellow and green as the flame is approached, while immediately about the light is a dark zone that separates it from the rainbow colored ring.

Less commonly patients complain during their prodromal stage of other chromatopsias; the seeing of colorless rings, or of stars. We suppose that these photopsias are caused by a slight œdematous opacity situated in the central part of the cornea. According to *Fuchs* this diffraction probably takes place in the deeper layers of the corneal epithelium. A conception of this phenomenon, met with in patients suffering from glaucoma, can be obtained by looking at a light through a glass coated with a 0.125% solution of erythrophlœin hydrochlorate, as suggested by *Treacher Collins*. Analogous color phenomena may also be observed in winter by looking through a window that is covered with frost.

It must be borne in mind that this chromatic vision is not characteristic of glaucoma alone. It happens not infrequently that patients who have complained of chromatopsia are sent to us by apprehensive physicians. Analogous symptoms appear even in simple conjunctivitis, when mucus, or a thin layer of blood, happens to lie on the cornea, and naturally disappear at once when these substances are removed. Colored areolæ may also appear about lights in commencing cataract, and even with the normal eye under suitable experimental conditions, but they do not appear paroxysmally.

Our patient has still other symptoms to tell us of. Together with the chromatopsia it seemed to him periodically as though he saw all objects through smoke, or as though a cloud was in front of his eyes. These periodical obscurations, which are met with in the prodromal stage of glaucoma, usually occur in connection with the chromatopsia. The vision need not be impaired; in bad cases the visual field is temporarily contracted. These obscurations are easily explained by the transient cloudinesses of the cornea. It may be concluded from the contraction of the visual field that the function of the retina itself may be impaired in bad cases.

Ciliary neuralgia is not uncommonly the most marked subjective prodromal symptom. Our patient tells us that formerly, before this severe attack of glaucoma, he suffered from analogous, though less severe pain. It is not without diagnostic interest to know that in some cases a severe trigeminal neuralgia may exist, to which is added later the development of glaucoma.

A distinct reduction of the light sense can be detected occasionally by means of *Foerster's* apparatus during the prodromal stage of glaucoma, at the time of the obscuration.

When we have to deal with patients who present symptoms suggestive of the prodromal stage of glaucoma we can also detect objective symptoms. Increase of tension is present, even in mild prodromal attacks, and sometimes we wonder how the vision can remain normal so long with a distinctly elevated intraocular tension.

The other characteristic features of glaucoma are not so marked during the prodromal stage. The pupil exhibits a tendency to dilate and a slowness in its reaction to light, but the inexpert must remember that the pupillary reaction is less extensive and prompt in elderly persons. A shallow anterior chamber arouses suspicion, but it must also be borne in mind that the anterior chamber becomes shallow in old eyes. One of the best evidences, in connection with the increase of tension, is a fine, smoky cloudiness of the cornea, but this is not always clearly demonstrable in the mild attacks of the prodromal stage. The testing of the sensitiveness of the cornea requires great care, according to my experience, in order to be able to draw from it positive conclusions as to the existence of a prodromal stage of glaucoma.

Two ophthalmoscopic symptoms may finally be mentioned. Occasionally, though rarely in comparison with the frequency of glaucoma, an arterial pulse may be observed on the papilla in the prodromal stage, when the branches of the central artery may be seen to become alternately pale and red, but only during their course over the papilla. We suppose that the central artery is so compressed by the increase of tension that the blood can no longer enter continuously, but that the greater quantity of blood impelled by the systole of the heart is still able to overcome the pressure, and so a sort of intermittent influx is produced. A symptom that is marked, and is characteristic of glaucoma, is that the veins of the papilla and retina are not bloodless, but on the contrary distended and tortuous. This sign of venous engorgement may be seen on the papilla itself, which appears to be greatly reddened. When we press upon the eyeball, and so cause an increase of tension in another way, the veins become smaller and the papilla pale, but in commencing glaucoma the venous hyperæmia exists with the increased tension and the arterial pulse, a sign that local changes have taken place in the vessel walls. The general practitioner however cannot be expected to know, recognize and discover these latter symptoms of a prodromal stage.

When elderly people complain of premature presbyopia, of photopsias, obscurations and ciliary pains, when the tension in each eye is thought to be increased, and there seems to be a suspicious tendency of the pupil to dilate, the prodromal stage of glaucoma should be taken into consideration.

119. Chronic Inflammatory Glaucoma and the Pathology of Glaucoma

This aged patient tells us that the disease in his eyes has developed very gradually. At times his vision has seemed to be rather bad, but he ascribed no special importance to this because he thought it a symptom of age. He has had slight pains about his eyeball occasionally, which extended down to his teeth, but he gave them no particular attention. The discovery that he could see only a very little with one eye and that he had a sensation of darkness on one side of the other led him to consult a physician in the hope that he could be helped with stronger glasses. He has changed his glasses frequently of late, always getting stronger ones.

Observation of the eye by daylight reveals a peculiar picture. The sclera appears strangely livid and the eye looks “dead.” Broad, dark blue, ramifying vessels course over the sclera and form a dense vascular plexus about the cornea. These vessels cannot be moved to and fro with the conjunctiva and are therefore the anterior ciliary veins, but while, under normal conditions, these veins run from behind forward and pierce the sclera near the margin of the cornea, they have formed in this case broad, ectatic branches, which have taken the form of a plexus.

The cornea does not appear perfectly transparent by focal illumination, and its surface exhibits minute defects of epithelium and irregularities. If we test its sensitiveness with a bit of cotton we find it to be distinctly diminished. The anterior chamber is extremely shallow, the iris and lens appear to be very close to the posterior surface of the cornea.

The pupil is dilated, is oval vertically, and reacts to light with extreme slowness in one eye and not at all in the other. A greenish gray reflex comes from within it. The iris is narrowed in consequence of the dilatation of the pupil, is shrunken above to a narrow edge, and no longer has a normal appearance. In some places we see gray spots, the relief and the fibrous structure are lost and it has evidently become very atrophic. The atrophic condition can be recognized still more distinctly with *Zeiss’* loupe.

The refractive media are still transparent and a typical glaucomatous excavation can be seen with the ophthalmoscope. The optic nerve is white, atrophic, deeply excavated; the vessels are pressed over to the nasal side and bend sharply over the margin in passing to the retina. About the papilla is a pale areola, the halo glaucomatosus.

Next we test the intraocular tension and find it distinctly elevated.

The reduction of the central vision and the contraction of the visual field, taken in connection with the deep excavation and the other objective changes in the eye, determine the diagnosis to be glaucoma.

The next question is: What form of glaucoma is this? The disease has developed spontaneously, insidiously, and in both eyes, without any other lesions acting as the cause of the increase of tension. It is therefore a primary glaucoma, of which there are three forms: the acute inflammatory, the chronic inflammatory and the simple. The latter we may exclude because the tension is increased, and because it is plain from the history of pains and obscurations that attacks of inflammation have occurred. The course of the disease is not that of an acute glaucoma, there has been no acute attack. Hence it is a chronic inflammatory glaucoma. Impress this clinical picture upon the memory. This is a disease that it is always well to bear in mind when we have to deal with the loss of vision of which old people complain.

The danger of this disease inheres in the fact that because of its gradual development its symptoms are often recognized too late, as in this patient. The longer a chronic inflammatory glaucoma exists the plainer become its symptoms; engorgement of the anterior ciliary vessels, immobility and dilatation of the pupils, shallowness of the anterior chamber, increase of tension and excavation of the optic nerve. Such eyes finally become blind, either very gradually, or during an acute attack.

The most essential clinical symptoms may be explained by the increase of tension, just the same as those of acute glaucoma. The principal avenue of escape for the blood from within the eye, the venæ vorticosæ, are compressed; consequently a compensatory engorgement is formed in the anterior ciliary veins. The vascular plexus about the cornea has been named the caput Medusæ. The increased tension is responsible for the advance of the lens and iris, the shallowness of the anterior chamber, the dilatation of the pupil, the insensitiveness of the cornea. It has caused the excavation of the optic nerve, the atrophy of the nerve fibres, the loss of vision, the contraction of the visual field, and finally the blindness.

Still glaucoma remains an enigma, for the question remains unanswered: Whence comes all this increase of tension? If we would try to reply to this question we must first investigate two things: we must know how the change of fluid takes place in the normal eye, if we would form an opinion concerning it under pathological conditions, and we must determine the pathological changes in the glaucomatous eye that govern the evident disturbance in the change of fluid.

An enormous amount of industry and labor has been expended on the study of the pathology of glaucoma, a labor that has merited better results than it is able to exhibit. I am convinced that the pathology of glaucoma predominates over a problem, to the solution of which it can contribute but moderately. The difficulties are these: It is inherent in the nature of things that very few eyes with commencing glaucoma have been examined pathologically, and yet we must depend on these primarily for knowledge of the nature of the disease. The most constant characteristics that have been found in the earliest stages of acute glaucoma are œdematous conditions in the posterior segment of the eye, in the papilla, retina and chorioid. In addition *Birnbacher* and *Elschnig* found a contraction of the veins that conduct the blood from the uvea; the venous emissaries of the venæ vorticosæ were infiltrated with cells and contracted.

Eyes in which glaucoma had existed for a long time have been examined far more frequently, and the lesions found may be studied from sections and drawings. Three cardinal signs constantly met with are: 1, changes in the blood-vessels; 2, excavation of the papilla; 3, changes in the filtration angle. None of the other lesions are so striking in comparison with these.

Glaucoma is a disease of persons in advanced life, and we know that a change takes place in the blood vessels in elderly people which causes them to lose their elasticity, to become worn out. Everyone thinks at once of arteriosclerosis, hence vascular changes have been sought for and found in glaucoma from the very beginning. Ever since the time of *Graefe*, attention has been called repeatedly to the frequent arteriosclerotic conditions in glaucomatous eyes. *Birnbacher* and *Czermak* found changes in the vortex veins, consisting of peri- and endophlebitic proliferations of cells which caused a contraction of the lumen. Others have been unable to find such an endophlebitis obliterans. Those who find these conditions are inclined to suppose that such a thickening of the veins in the principal outlet for the intraocular blood may produce a venous engorgement and an increase of tension. Vascular changes have also been found, though again not uniformly, in the second most important outlet, the ciliary veins and *Schlemm's* canal. *Schlemm's* canal is sometimes completely filled with blood, sometimes partially obstructed, sometimes totally occluded. Pigment cells may be met with in its wall and in its lumen.

The conditions in the chorioidal vessels are likewise full of contradictions. Marked sclerotic thickenings of their walls are found in some eyes, nothing of this nature is present in others. Sometimes an endarteritis is met with that has produced a total obliteration. In some cases the vessels of the chorioid are dilated, in others they are atrophic. On the whole we must agree with *Bartels* that the changes in the vessels of the chorioid represent only varying degrees of the sclerosis which we meet with in age without glaucoma.

The same condition confronts us in the vessels of the iris. When normal they are somewhat thicker and more rigid physiologically in age, and we also meet with proliferations of the adventitia. Increased fullness alternates with atrophy of the iris and hyaline degeneration of the vessels. The vessels in the ciliary body are likewise in part dilated, in part contracted. We sometimes meet with proliferation of the endothelium, and again with perfectly normal construction. A comparatively constant anatomical condition is the dilatation of the anterior ciliary veins, but it must not be forgotten that they too have been found stenosed. The conditions in the anterior and posterior ciliary arteries are too inconstant to permit any conclusions to be drawn from them.

Greater importance is to be ascribed to the changes in the retinal vessels. Endarteritis obliterans and hyaline degeneration of the walls have been found repeatedly, and made answerable in part for the glaucoma. A homogeneous wall of the sclerosed retinal vessels, without nuclei, should be typical of glaucoma, but this statement is not in harmony with the actual conditions. We meet with fibrous thickenings, proliferations of the intima, and normal vessels. Thromboses in the central vein have been noted in certain forms of the disease, but the question is: Do the diseases found in the vessels present anything characteristic or specific of glaucoma? Can it be recognized from a microscopical examination of these vessels that glaucoma was present? This question has been answered again in the negative very recently by *Bartels*, and, in my opinion, the reply should be most emphatic.

We will turn from the blood vessels to the glaucomatous excavation, which was demonstrated anatomically for the first time by *Heinrich Mueller* in 1856. In contrast to the vascular changes this is the symptom of the pathology of glaucoma which is characteristic of the disease. It will be noticed first that the lamina cribrosa is strongly pressed backward, thickened anteriorly and compressed. There is a deep cup in the head of the optic nerve, its bottom formed by the lamina cribrosa, its lateral walls by the sclera. Traces only of the original tissue of the papilla are present, consisting of neuroglia, atrophic nerve fibres and drops of myelin, which form a thin layer over the bottom of the excavation. Remains of atrophic nerve fibres also rise along the steep walls and bend over the sharp "neck." The vessels are pressed together in the nasal portion. I wish to call particular attention to the extreme thinness of the retina at the sharp margin of the cup over which the vessels pass. It can be seen in some preparations that peculiar fissures and cavities may be formed in the papilla during the development of the excavation. I would not call special attention to this were it not that it has been named by *Schnabel* "cavernous disease of the optic nerve," and was considered by him to be a lesion characteristic of glaucoma alone, but there is no doubt that cavities are met with in the papillæ of eyes that are not glaucomatous, as has been shown by *Schmidt-Rimpler*, *Schreiber*, *Axenfeld*, *Stock* and others.

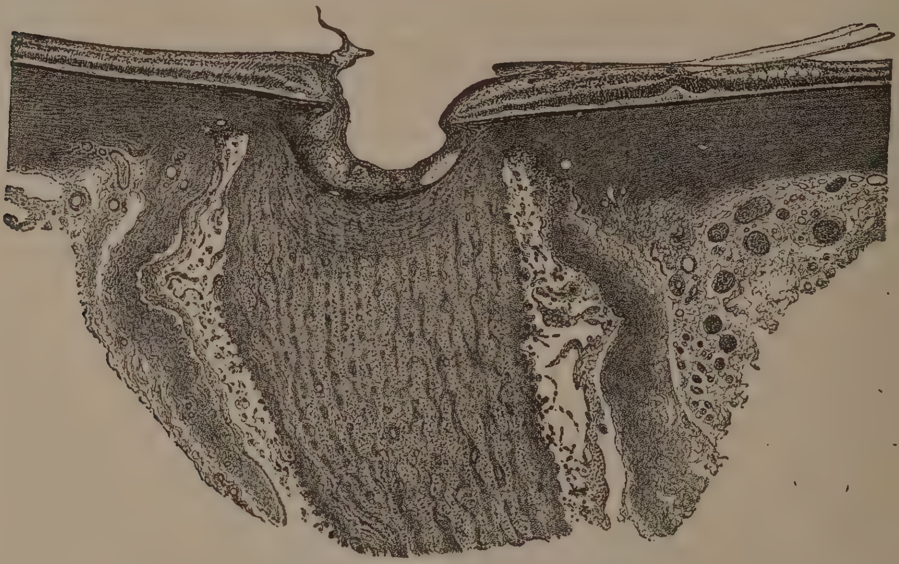

FIG. 99.—Glaucomatous Excavation.

We come now to the third and most important cardinal symptom of the pathology of glaucoma, the occlusion of the sinus of the anterior chamber. It is here, between the ciliary body, iris, and cornea that the intraocular fluid leaves the eye and flows into *Schlemm's* canal, a circular vein. This space, known as *Fontana's* space, is the principal outlet of the aqueous. *This outlet is occluded in extremely many glaucomatous eyes.* It is evident at once that the occlusion of such an outlet may, under certain circumstances, be fatal to the eye.

This occlusion of the filtration angle may be produced in various ways. In numerous cases we find the root of the iris pressed against and adherent to a considerable extent of the posterior surface of the cornea. In other cases only the posterior part of the sclerocorneal bundles of fibres are covered by the iris, so that only a part of *Fontana's* space is obstructed. This adhesion of the root of the iris to the corneoscleral margin, whether of greater or of less extent, evidently may arise in two ways: a primary inflammation, which results in adhesion, may take place in the sinus of the anterior chamber, or we may imagine that the root of the iris is pressed from behind against the cornea and then grows adherent. Numerous investigators, among whom *Czermak* and *Birnbacher* are prominent, have found signs of adhesive inflammation, newly formed tissue, in the sinus of the anterior chamber, while *Ad. Weber* thinks that the root of the iris is pressed forward by the venous engorgement of the swollen ciliary processes.

There is still another way in which the outlet may be closed in glaucoma.

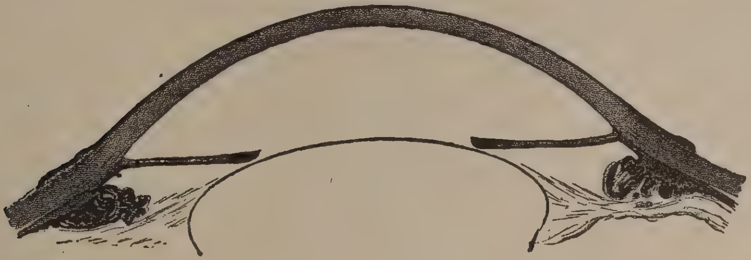

Fig. 100.—Occlusion of the Sinus of the Anterior Chamber in Glaucoma.

The sinus of the anterior chamber may be perfectly open and the obstruction be in *Schlemm's* canal. Stenoses, due to inflammation in its neighborhood, which obstruct the outflow, are met with. This occlusion of the filtration angle, in one form or another, is the rule in glaucomas that have lasted long.

Still there are exceptions to this rule. I have a properly prepared eye with glaucoma simplex in which the filtration angle is open. In such cases the difficulty in filtration must be due to other causes, such as atrophy, or indurative inflammation of the trabeculum sclerocorneale. These are the most important and most utilizable facts to be drawn from the pathology of glaucoma to account for its origin.

Among the lesions in the other tissues of the eye are œdematous swellings to be found in the retina, especially near the papilla, in recent cases of glaucoma. In cases of long standing the retina is finally transformed into a thin, atrophic, fibrillary membrane. It is worthy of note that the layer of fibres and the layer of ganglion cells are the first to suffer, while the outer layers may be preserved for a long time. According to the researches of *Schreiber*, quite a brief pressure suffices to give rise to a formation of vacuoles in the ganglion cells, and to an ascending atrophy in the layer of nerve fibres.

We look upon the halo glaucomatosus as a simple atrophy of the chorioid in the vicinity of the papilla. Cellular infiltrations, signs of a chronic inflammation, are frequently to be found in this membrane. The conditions indicate the presence of a vascular hyperæmia with exudation in the beginning of an inflammatory glaucoma, which terminates in atrophy.

The ciliary processes are surcharged and swollen at first; later they become atrophic and are frequently elongated. When a great extent of the iris is adherent to the cornea at the sinus of the anterior chamber we find a large interspace between the apices of the ciliary processes and the iris, and then we can no longer say whether the swollen ciliary processes pushed the root of the iris forward or not at the commencement of the disease.

In old glaucomas we often find a new formation of vessels between the epithelium of the cornea and *Bowman's* membrane (pannus glaucomatosus).

The œdema predominates in acute glaucoma and inflammatory infiltrates occur in older cases. We also meet with keratitis bullosa, in the production of which trophic disturbances of the nerves and transudations from the marginal network take part.

Priestley Smith called attention to an unusual size of the lens in glaucomatous eyes, which narrows the circumlental space, a factor to which we shall return in the study of the origin of glaucoma. On the other hand, a marked increase of the lens itself has not been confirmed, although the contraction of the perilenticular space has generally been recognized.

The sclera has likewise been studied with varying results. *Czermak* and *Birnbacher* found a lack of nuclei and atrophic changes in the inner layers of the sclera, but we know that under physiological conditions this membrane becomes more rigid with age and loses its elasticity. The changes in the vitreous are, in my opinion, most important with reference to the alterations in the change of fluid, but they are not sufficiently susceptible of a single interpretation.

So much concerning the pathology of glaucoma. We can trace the essential clinical symptoms to the increase of tension, but the origin of the latter cannot be explained by the pathology, for the initial stages have not yet been sufficiently investigated, and we do not know which of the generally well marked lesions were primary and which were secondary, which was the cause and which the result of the increased tension. Even if we were better acquainted with that anatomical basis of the initial symptoms, who could tell what started the trouble from the study of the anatomical preparations? Many interpretations have been given to the anatomical conditions. Very many investigators have toiled over this subject, an extremely large number of eyes have been examined microscopically, important facts have been learned, but pathology has not explained the nature of glaucoma. The solution of this problem will, I think, scarcely be attained in this way, for the disturbances of cell life on the one hand, and the relations of the eye to the entire organism necessary for the intraocular change of fluid on the other, permit little if anything to be gathered from the microscopical picture. We must therefore try to obtain help from the physiology of the change of fluid in the eye, if we will get at least a little nearer to understanding the clinical picture of glaucoma.

Although the pathology of glaucoma is unable to teach us the origin of this disease, yet gain is derived from it in regard to treatment. Of the three cardinal symptoms in the glaucomatous eye, the changes in the blood vessels, the excavation of the optic nerve and the alterations in the filtration angle, the third alone is amenable to treatment. We instill meiotics, in order to contract the dilated pupil and to draw the iris away from the filtration angle, or we employ operations to reopen the occluded outlet at the angle of the anterior chamber, which may be witnessed in the operating room.

120. Glaucoma Simplex and the Conservative Treatment of Glaucoma

The disease of the eye exemplified in this patient is diagnosed in almost all cases by the recognition of a glaucomatous excavation of the optic nerve with the ophthalmoscope. We know that primary glaucoma appears in three different forms, as acute inflammatory, chronic inflammatory and simple. This patient complains only of a gradual loss of vision and—this is worthy of note—of no other special trouble.

Both eyes are pale, the anterior ciliary vessels are not particularly tortuous on the sclera, the cornea is perfectly transparent and normally sensitive, no shallowness of the perfectly normal anterior chamber can be detected, the pupil is not in the least dilated, it reacts promptly and there is no gray green reflex to be seen in it. In a word, everything in the anterior segment of the eyeball is entirely different from what is met with in both of the other forms of primary glaucoma. How is it possible that this can be glaucoma? Two principal symptoms of that disease are certainly present, the gradual loss of vision and the excavation of the papilla, but where is the increase of tension?

It is a most important fact in glaucoma simplex that even under prolonged observation a pathological increase of the intraocular tension frequently cannot be demonstrated. None can be felt in this case. How was the glaucomatous cup formed in the optic nerve, which we still consider to be a pressure excavation? Three pathological facts furnish an explanation.

1. There are cases of glaucoma simplex in which a pathological increase of tension appears early in the course and then ceases.

2. Careful clinical observation has proved that temporary increases of tension occur at times in the course of the disease. The case is then one of glaucoma simplex with intermittent fluctuation of tension. Such patients may complain of prodromal symptoms, periodic obscurations with or without seeing the colors of a rainbow. If the tension is tested regularly every two or three hours with a tonometer these fluctuations of tension may often be brought out quite distinctly. The tension fluctuates between the upper limit of normal to + 1.

Such fluctuations of tension in glaucoma simplex may sometimes be ascribed to exciting factors, such as psychic excitement, disturbed sleep, menstruation, or congestions at the time of the climacteric. These increases of tension are often most marked just before going to sleep and on awaking in the morning. Such temporary, frequently repeated increases of tension are able to produce gradually an excavation of the papilla.

3. Even when no pathological increase of tension can be detected by prolonged observation the development of the excavation is not inexplicable. I have already pointed out that the intraocular tension fluctuates considerably under physiological conditions. When we are dealing with an eye suffering from glaucoma simplex we do not know how high, or more correctly perhaps, how low its tension was in youth. If its tension has increased with age only so far that it does not exceed the highest limit of physiological tension, it may still have been a considerable increase, sufficient to produce very gradually a pressure excavation.

The course of glaucoma simplex is such that the vision sinks quite gradually to blindness. Both eyes are attacked, as a rule, though it may be at different times. Permanent confinement of the disease to one eye is very rare, but occasionally happens, a respect in which this contrasts with the inflammatory forms of glaucoma. Cases are also met with in which considerable increases of tension occur from time to time, with all the signs of an attack of acute glaucoma (ciliary injection, shallowness of the anterior chamber, cloudiness of the cornea, and pain). These are said to be cases of glaucoma simplex with intermittent inflammations. They may pass over permanently into the condition of chronic inflammatory glaucoma, but in the great majority the excavation of the optic nerve is the only symptom from which this disease can be recognized. The glaucomatous excavation can be studied best in such eyes, because the refractive media are clear.

The cardinal symptoms in a marked case of glaucomatous excavation are as follows: The papilla is pale, bluish, or greenish white and there is a deep cup of the head of the optic nerve that extends up to its margin. The gray dots that mark the lamina cribrosa may be seen at its bottom. The vessels do not come from their usual place, the middle of the papilla, but from its inner margin, and they bend sharply at the place where they pass over its edge to the retina. It can be recognized best from the vessels that the lamina cribrosa has been driven backward, and that a deep cup has been formed. The course of the vessels over the entire papilla can be seen only in the inverted image. In the upright image, if the eye is focussed on the margin of the excavation, portions of the vessels have indistinct outlines, because they lie farther back, at another level, and are out of focus. The papilla is surrounded by a yellowish border, the halo glaucomatosus. We will reserve the more accurate study of this excavation and its differential diagnosis, which is not always easy, for the course in ophthalmoscopy, to which we will have our patient return.

The following points may be noted in regard to the reduction of the central vision and the contraction of the visual field in glaucoma.

The contraction of the visual field starts from the periphery on the nasal side and usually above. I have already called attention to the fact that the outer portions of the retina are the first to become functionally incapacitated. The vessels with the nerve fibres running over them are usually pressed over to the nasal side, so the nerve fibres that run in a temporal direction are generally dragged upon. Moreover, this layer of nerve fibres which passes over the temporal half of the optic nerve is thinner and more easily compressed, and the blood supply to the temporal side of the retina is not as good as that to the other side. These conditions explain why the contraction of the visual field begins in the nasal portion in 50 or 60% of the cases. Paracentral scotomata are occasionally met with, especially near the blind spot.

Another point characteristic of glaucoma is that a narrow sector of the visual field, extending outward from the point of fixation, may be preserved for a long time.

The long persistence of the color sense is striking, even when the field is very contracted; even the ordinary arrangement of the color sensation, green within, then red and then blue, is preserved in the small field of vision.

We come now to the most important question, the treatment. What can we do for our patient? We have at our disposal a conservative and an operative treatment, sometimes a combination of both, for primary glaucoma. I have intentionally associated this consideration of the treatment of primary glaucoma with this case of glaucoma simplex. To operate, or not to operate, that is the question.

Sometimes it is very difficult to decide how to treat an individual case of primary glaucoma. It might perhaps be expected that ophthalmology should long ago have determined whether this form of glaucoma should be operated on and that form treated conservatively, but until 1856 this was considered an incurable disease. Then came *Albrecht v. Graefe* with his discovery that the intraocular tension in glaucoma is reduced by an iridectomy. It was right that this blessed discovery should be greeted enthusiastically by all, for it has enabled thousands of men to preserve for years their gravely threatened vision, but there was an excusable error in the enthusiasm which led the students and admirers of *v. Graefe* to proclaim iridectomy as the remedy for glaucoma. Iridectomy does not affect the nature of this disease, it only removes a link from the chain of glaucomatous symptoms by the elimination of a mechanical factor. This is a necessary corollary to the following clinical fact: in the majority of cases of glaucoma in which a permanent reduction of the intraocular tension has been effected by this operation, the diminution of the visual field and the loss of vision continues and cannot be stopped. Other things that appertain to the nature of the morbid process, such as affections of the optic nerve, run their courses in glaucomatous eyes independently of the hypertony. The longer the patients operated on can be kept under observation, the smaller becomes the number that can be called permanently cured. All soberly thinking ophthalmologists share this conviction. I will quote, for example, the opinion of *Logetschnikoff* which he formed from his abundant experience for years in about eight hundred iridectomies and two hundred and eighty-four sclerotomies. "In consequence of my observation for many years of a large number of patients on whom I had performed one or the other of these operations, I have become convinced that the operation delays the glaucomatous process for a longer or shorter time, but does not cure it. Exceptions are extremely rare and are met with only in two classes of cases: the rare cases of acute glaucoma which are operated on early, and the so called prodromal glaucoma, in both of which no excavation of the optic nerve, and in general no permanent glaucomatous changes are present, when the process tends to be brought to a standstill either spontaneously, or under treatment with eserin. These extremely rare cases of cure stand in contrast to others in which the operation does decided harm and the vision sinks rapidly to blindness after it has been performed." Such a recognition of the limits of our operative skill does not detract from the great credit due *A. v. Graefe*, but places the former simply in the light of truth. It does not lessen the great importance of the operative treatment of glaucoma, for if we are able only to save the vision for years, to hinder the development of this frightful disease, to delay the onset of blindness by our operations, the therapeutic accomplishment is one of which ophthalmology may well be proud. It can be readily understood that the present generation of ophthalmologists, who do not regard the accomplishments of *v. Graefe* through a dense cloud of incense, no longer have immediate and indiscriminate recourse to the knife, as in the days when everything was hoped for from the operation for glaucoma. The fact is that both the physician and the ophthalmologist turn, and should turn, first to the medicinal treatment in every case. Whether the case is one of prodromal symptoms, of a serious acute attack, of the chronic inflammatory form, or of glaucoma simplex, we always try meiotics first.

Two remedies are employed: physostigmin or eserin, in a 0.5 to 1% solution, and pilocarpin, in a 1 to 2% solution. Both drugs have won a conspicuous place in the fight against glaucoma. The value of meiotics in the treatment of this disease was pointed out by *Laqueur* and *Ad. Weber* in 1876.

Numerous experiments concerning the action of meiotics upon the normal eye have shown that a slight increase of the intraocular tension is reduced by them. This effect is ascribed to a contraction of the intraocular vessels and to a diminution of the secretion of the intraocular fluid. Such an effect is exactly what is desired in glaucoma. The reduction of tension in this disease by eserin was first established by *Laqueur*. Eserin and pilocarpin diminish the size of the pupil through a spasmodic contraction of the sphincter, draw the periphery of the iris away from the sinus of the anterior chamber, and so reopen this channel for the outflow of the aqueous. This factor plays a large part in the treatment of glaucoma with meiotics.

The best way to learn the effect of these remedies is on one's self; a way which is also excellent in that it enables us to appreciate the annoyances associated with their action, and to sympathize with patients when they complain.

A few minutes after the instillation of a drop of eserin into an eye slight spasms are felt, which continually grow stronger and are excited by the contractions of the ciliary muscle. The eye naturally becomes temporarily nearsighted, sometimes a severe pain is felt whenever an attempt is made to look at a near object, and the onset of meiosis is soon demonstrated. These meiotics owe the favor shown them to the ease with which they may be used and the immediate effect, which can easily be demonstrated on the normal eye as well as in some forms of glaucoma, but ophthalmologists are not agreed in the estimation of their value.

Some maintain not only that meiotics arrest the glaucoma and can be used as adjuvants to the operative treatment, but that they as such should supplant the operations, because they are actual remedies for the disease. Among these are *Pflueger*, *Cohn*, *Schleich*, *Koenigshoefer* and *Bjerrum*. This view has logically led some ophthalmologists to regard an operation as a last resort.

Others hold a view diametrically opposite, that time is often wasted with meiotics and that vain hopes of a cure without an operation are held out to patients. Among these are *Schweigger* and *Groenouw*. *Schoute* took the trouble to tabulate the views of ophthalmologists concerning the treatment of glaucoma, and we obtain from his table a sad picture of the distraction of opinion concerning this important question. When opinions are so divided how can the physician decide how far he may go with the medicinal treatment of glaucoma, and when he should advise operative intervention? This question interests the ophthalmologist as well, for he cannot form an opinion from his experiences accumulated in a rambling way, which are perhaps hazy from the lapse of years, but must wish that the clinical observations accumulated for decades by our predecessors enabled him to take a firm ground in regard to this matter, on which he could act without harm to his patients. Unfortunately the material furnished in our literature is not calculated to facilitate such a search for a firm foundation. This is due in great part to the difficulty and complexity of the entire problem of the treatment of glaucoma. If our predecessors had thought more of the future, and at the same time had considered that a final judgment concerning the treatment of this disease would not be possible for decades, if they had rather facilitated the work of criticism of later generations, we would be better conversant than we are with the true value of treatment. It must be possible, in our civilized country, that we should be better informed than we are concerning the fate of eyes that have been treated for glaucoma. In 1856 *v. Graefe* discovered the effect of iridectomy; during more than half a century the operation has been employed in many thousands of cases. Since 1876 meiotics have been used in numberless cases for the conservative treatment of glaucoma. What has become of all the eyes? Where are the statistics that may show the final results? The number of cases in which the results of treatment have been observed for one or more years is insignificant in comparison with the enormous number of glaucomas that occur, and the number that have been watched for a decade or longer are rarities in comparison with the total number. This is partly because glaucoma is usually met with in the advanced years of life and the patients die, but the majority of patients who have been treated evidently leave their first physician after a time, it may be after years, because the result of his treatment has not been satisfactory. This is a sure sign that the improvement lasts only for a time, in the majority of cases, and that then a contraction of the visual field and a loss of vision supervenes. Few statistics are calculated from the point of view that many years of observation are necessary to the passage of judgment. We must lay hold of these, and when, by their aid, we compare the results of the operative treatment with those of the conservative we unquestionably obtain the following knowledge.

Neither the operative, nor the conservative treatment can cure glaucoma, in the true sense of the word. The problem of its cure is by no means solved. In our efforts to combat the disease we cannot adhere to either method, but must use both. Conservative treatment should be tried first in every case of primary glaucoma, but, before entering upon this, we will consider the results of the operative treatment as presented in the table compiled by *Schmidt-Rimpler*. It must be remembered, however, that the actual, permanent, curative results are considerably less than is shown by the table. Not only is the permanent benefit from the operative treatment limited, but experience has proved that in one class of cases a deterioration of vision is directly caused by the operation. We will learn these disadvantages and complications at another time. All these conditions impose on us the serious duty of considering maturely whether we must operate, to seek urgent indications for the operation, and always to determine first whether we cannot succeed without it.

AUTHORS	Glaucoma Acutum			Glaucoma Chronicum			Glaucoma Simplex		
	Number	Improvement or Arrest	Deterioration	Number	Improvement or Arrest	Deterioration	Number	Improvement or Arrest	Deterioration
Haab	31	24	7	37	21	16	76	54	22
Sattler	6	5	1	9	5	4	10	6	4
Mellinger	23	18	5	28	15	13	24	6	18
Saemisch	21	19	2	30	19	21	64	23	41
Rossalino	33	28	5	48	26	22	41	24	17
Wygodski	37	30	7	147	74	73	129	20	109
Koster	16	10	6	16	7	9	17	5	12
Schmidt-Rimpler	....	....	....	....	....	....	10	3	7
Axenfeld	....	....	....	....	....	....	10	2	8
Démétrides	....	....	....	....	....	....	152	11	141
Krukow	....	....	....	....	....	....	17	4	13
Nuel	....	....	....	....	....	....	8	....	8
Schirmer	....	....	....	....	....	....	5	2	3
Schleich	....	....	....	....	....	....	102	16	86
Laska	....	....	....	....	....	....	39	19	20
Peillas	....	....	....	....	....	....	67	54	13
v. Hippel	....	....	....	....	....	....	57	30	27
Total....	167	134	33	315	167	158	828	279	549
Percentage....	....	80	20	....	53	47	....	33.7	66.3

Results of the Operative Treatment of Glaucoma

On the other hand consideration of the results obtained by the conservative treatment of glaucoma shows that this also has certain limits. A number of cases have been reported in which prodromal attacks were controlled for more than a decade, and in which the course of glaucoma simplex was delayed for ten or more years, but it has not been proved that the conservative treatment is the only remedy in a single form of glaucoma. *Schleich* observed an advance in 61% of his cases of glaucoma simplex, an arrest in 39%. Other statistics tell the same story. There can therefore be no doubt that we cannot adopt one method of treatment. The question always remains to be answered, in the use of meiotics, whether we are not missing the correct indication for an operation. All these discords are convincing that the choice of the form of treatment appropriate for the given case is a most important and responsible duty. A universal prescription cannot be given, because the forms of glaucoma differ too greatly, and because both mild and malignant cases of the same form are met with. We can only try to lay down certain principles for guidance. The peculiar nature of each individual case must be taken into account, for if there is a disease in which individualization is imperative, that disease is glaucoma.

The general practitioner has the easier time, as he uses only the conservative treatment. If a patient comes to him with an attack of acute glaucoma he should immediately instill some drops of a solution of pilocarpin or eserin (1%) and send him to some specialist. He should do the same with a patient who has the prodromal symptoms, as well as when the diagnosis of chronic inflammatory glaucoma has been made, or is suspected. In glaucoma simplex the general practitioner awaits instructions from the clinic, or the specialist, in regard to the management of the case. The general practitioner throws the responsibility of the treatment of a case of glaucoma upon the specialist, yet he has important duties to perform in the constitutional treatment, when the patient has been referred back to him. I stated, on a former occasion, that many lesions outside of the eye had been held responsible for the development of glaucoma, and the final cause is doubtless situated in the general organism. Persons with a gouty diathesis and nervous individuals are predisposed to this disease. An attack may be brought on, or the disease made worse, by nervous excitements, or such troubles as circulatory disturbances during the climacteric. It is therefore the duty of the general practitioner, as it is our custom, to investigate the entire organism of a patient predisposed to, or suffering from, glaucoma, and to regulate his entire mode of life. When possible, an entire syllabus should be worked out for such people, in which naturally the social conditions must be taken into account. I can mention only a few fundamentals: Regulation of the diet, with special care for easy and regular movements of the bowels; control of the metabolism so far as possible; constitutional treatment according to the general condition; when there is a predisposition to gout, alkaline mineral waters, springs containing lithium; when there is arteriosclerosis, preparations of iodine; when there are nervous indications, quinine, bromine and tincture of valerian; avoidance of congestion of the head; restriction of alcohol and tobacco; no straining in a stooping posture, no hot baths, preferably warm foot baths.

It may be asked whether there is not a general treatment which is able to obviate the disturbances in the change of fluids in the eye from within the organism. Many attempts have been made to discover such a treatment, but thus far they have been fruitless. For example, a rise in the blood pressure has occasionally been striven for (*Zimmermann*), although we used to fear the rise of blood pressure in glaucoma. Others, like *Cantonnet*, and *Lapersonne*, have tried to secure an increased excretion of lymph from the eye by large doses of common salt, when the renal functions were good, or, when these functions were bad, by saltless food, lactose, rice and purgatives. In addition to the regulation of the general organism such patients are thus prescribed a certain eye diet.

All unnecessary irritation of the eyes is to be avoided, because the optic nerve and retina are endangered. To work, or even to remain, in our modern intense illumination is harmful. The chemically acting ultraviolet rays have been demonstrated to be particularly injurious to the retina, so we order suitable protecting glasses for our patients. For this purpose we have at our disposal various kinds of glasses which arrest these rays, the best of which are the

Hallauer glasses, and the euphos glasses of *Schanz* and *Stockhausen*, but the old blue and gray glasses are also useful.

Above all, patients with glaucoma should be advised not to overstrain their eyes with near work. On the other hand, it is not advisable to prohibit all reading and writing, which should be rendered as easy as possible with proper glasses.

It has been repeatedly observed that the prodromal attack is overcome best through the accommodation, by the reading of fine print. A contraction of the pupil accompanies accommodation and this opens wider the sinus of the anterior chamber. It has also been supposed by *Kuesel* that the contraction of the ciliary muscle influences favorably the filtration of the aqueous, by the fibres that extend to the ligamentum pectinatum.

Massage of the eye in various ways has been recommended; pressure massage with the hand, vibratory massage with certain instruments, pneumatic massage with an airpump, but I consider all these measures to be very hazardous when applied to a glaucomatous eye, and cannot recommend them. I think just as little of galvanization of the sympathetic, the negative electrode placed on the nape of the neck, the positive on the anterior margin of the sternocleidomastoid, which is occasionally recommended.

Anodynes are frequently needed in absolute glaucoma and during the attacks of pain; in such cases we may instill cocain and pilocarpin combined, scopolamin and dionin. Good sleep at night is to be secured by regular, though not excessive, pleasure walks and, when necessary, by means of one of the numerous modern hypnotics.

In the therapeusis of individual cases of glaucoma I try to manage so as to avoid an operation if possible. I treat a commencing glaucoma in the prodromal stage with obscurations and rainbow colors, with eserin or pilocarpin, daily instillations morning and evening, under all circumstances. Three things are then possible: 1, a pronounced glaucoma does not develop, in which case an operation would have been unnecessary; we know that the operation is not devoid of danger, but is directly injurious in some cases; 2, the prodromal attacks become rather less frequent and a chronic inflammatory glaucoma develops with its indications; 3, the number or intensity of the prodromal attacks increases, showing that the meiotic is unable to regulate the tension. Then an operation is necessary.

In an attack of acute glaucoma we likewise prescribe eserin at first, but we do not wait long, for days as has been recommended, but decide upon our course from the following indications: If, according to the history, this is a first attack, and it is therefore to be expected that no excavation has been formed as yet in the optic nerve, we perform an iridectomy, for the fact has been established that in the first attack of acute inflammatory glaucoma iridectomy produces the best and most permanent result. Sometimes its performance is very difficult, because the anterior chamber is so shallow, but numerous observations have shown that the excision of a portion of the iris to its periphery is not so absolutely necessary in acute as in chronic inflammatory glaucoma. It is to be understood that the operator will make every effort to extend the iridectomy as far as possible toward the periphery, but in this case an incomplete excision causes a retrogression of the increased tension. An iridectomy must also be performed immediately in the rare cases of fulminating glaucoma, in which perception of light seems to be gone from the start. No time must be lost in such cases, for it is only in a few that any vision can be saved.

If the attack of acute glaucoma is not the first, if it is probable that the optic nerve has suffered already and that the visual field is contracted, we adopt the same measures of treatment as in chronic inflammatory glaucoma, in which it is generally acknowledged that the results of operative intervention are by no means so good as in the acute form. We may suppose that in the latter the retina and optic nerve recover readily after the tension has been reduced. In chronic inflammatory glaucoma, on the contrary, we have three things to fear.

One is that the operation may be directly injurious, and everyone will subscribe to the position taken by *Schmidt-Rimpler* in the latest work on glaucoma. "It may be understood that a careful physician, especially when he is dealing with the sole remaining eye, which still retains a comparatively good vision, weighs very carefully the question whether he shall have recourse to an operation."

A second is that the atrophy of the optic nerve will advance and the vision be lost, as happens in many cases of chronic inflammatory glaucoma after the removal of the increased tension by the operation. The patient is quite apt to consider the operation responsible for this.

In the third place it must be remembered that the final result in chronic inflammatory glaucoma is not as good as in the acute form. Therefore I do not approve of the stand taken by many other ophthalmologists, that we should operate as early as possible on patients with glaucoma inflammatorium chronicum, I believe on the contrary that we should first try to save what vision they have by means of meiotics. Nevertheless we should not delay the operation until nothing can be hoped for from its performance. We test the vision and visual field of such patients regularly and estimate the tension tonometrically. When, in spite of all conservative treatment, it is evident that the vision is failing, that the tension is not to be permanently regulated, and especially when the patient is convinced that no more progress is to be made in this manner, we proceed to operate, on one eye at first.

The iridectomy must be performed with the greatest possible exactitude in chronic inflammatory glaucoma. If the anterior chamber is very shallow and the iris is very atrophic and narrow, I perform a cyclodialysis as an adjuvant and preliminary operation and then, after a few days, when the conditions in the anterior chamber are more favorable, but the regulation of the tension is not yet satisfactory, I perform the iridectomy.

The results of operative treatment are so bad in glaucoma simplex that conservative treatment is to be preferred under all circumstances. The condition of the intraocular tension is tested regularly for several days; tonometry is particularly valuable for this purpose. An operation is justified only in cases with pronounced hypertony, for it has been proved that, in this form, a malignant course may be inaugurated more readily by an iridectomy than by any other procedure. When some operation must be performed sclerotomy or cyclodialysis should be chosen.

121. Glaucoma Absolutum and the Differential Diagnosis of Glaucoma

This patient presents in each eye an absolutely typical and peculiar clinical picture. The most striking characteristic is the bluish white color of the sclera, resembling porcelain, upon which are numerous, extremely thick, bluish red vessels, the greatly distended ciliary veins and their branches. The entire cornea is surrounded by such a wreath of vessels, the caput Medusæ.

There is no very sharp differentiation between the cornea and the sclera, the groove between them is abolished, and the entire limbus has a peculiar bluish color. The cornea itself looks opaque, though the opacity differs in various places. In part the cornea looks glassy, little irregularities can be seen in its surface and its sensitiveness is lost. The anterior chamber is completely abolished, the pupil is dilated ad maximam, the iris is quite narrow and seems to be pressed against the posterior surface of the cornea. It can be perceived that the iris is atrophic from its gray appearance; often it contains small, isolated spots. The eyeball is as hard as a stone and totally amaurotic. These symptoms give us the diagnosis: glaucoma absolutum.

We understand by absolute glaucoma that stage of the disease in which the vision is totally lost. When the refractive media still remain fairly transparent we obtain from the dilated pupil the characteristic gray green reflex of the senile lens, and we may see with the ophthalmoscope the glaucomatous atrophy of the optic nerve with its excavated papilla. The blood vessels are very small. Retinal hemorrhages and pigment changes in the fundus may also be observed.

The refractive media are no longer clear in this patient and we will examine rather more closely the opacity of the cornea. We designate the corneal changes met with in absolute glaucoma as glaucomatous degeneration. This appears in various forms.

In this case the cornea is stippled, partially deprived of epithelium and an opacity has developed, as usual in the lower segment, into which superficial vessels from the conjunctiva proliferate. It is thought that the lower part of the cornea, which moves upward when the eye is closed, is particularly apt to be injured during sleep.

In other cases the epithelium becomes detached in the form of bullæ, which burst and leave irregular tags hanging from the surface of the cornea (keratitis bullosa). Ulceration occurs readily in such an insensitive cornea. Peculiar ribbonlike, whitish and yellowish opacities are also formed in the region of the palpebral fissure.

The origin of these glaucomatous degenerations will seem comprehensible at once when it is borne in mind that such a cornea must be less capable than it should be of resistance to external and internal injuries, in consequence of the disturbance in its lymph circulation and nutrition, as well as of its insensitiveness.

The opacities of the lens are of particular interest for various reasons. In this patient the lens is totally opaque, so that no vision into the eye is possible. All of these changes in absolute glaucoma indicate that the production of blindness is not the final act in this tragedy. Such patients have many subjective troubles and objective lesions.

Such an eye often reddens and lacrymates without any obvious cause, while attacks of severe drawing pains still continue to occur in the brow and head. This painful stage of absolute glaucoma passes away and the patient is freed, for a while at least, of these grievous pains. In addition to these attacks of pain, or even without them, peculiar clouds of light appear to some patients with glaucoma in this stage, which naturally give rise in many cases to the vain hope of a return of the lost vision. Sometimes this sensation that it is brighter in front of the eye appears at certain hours. *Rydel* conjectures that the varying sensations of light and dark are dependent on the fluctuations of the intraocular tension. His idea is that if the circulation in the retinal arteries should be completely arrested by the increased tension the sensation of darkness would be caused, and then, when the tension relaxed, the influx of blood would again stimulate the retina.

The eyeball may either atrophy, or become enlarged. Phthisis may come on quite gradually; the vitreous contracts, the retina becomes detached, the cornea diminishes in size, the shrunken lens with the iris lies closely against it, the eye has become soft. Phthisis may also follow the perforation of a corneal ulcer, when serious intraocular hemorrhages may take place, some of which have been reported to even endanger life.

When the eyeball becomes enlarged we usually find staphylomata between the cornea and the equator. The sclera grows thinner and thinner and the bluish looking uvea shines through. In addition to these partial ectasiæ the eyeball may be enlarged in all of its diameters. Recurrent hemorrhages and attacks of pain are quite apt to be present in such eyes. The final result of glaucoma is extremely sad and, under such circumstances, enucleation is a blessing.

A knowledge of the differential diagnosis of glaucoma is of the greatest importance to the general practitioner, because a timely, correct diagnosis may save many an eye, while an incorrect one, or one made too late, may spoil everything. The general practitioner must work hand in hand with the ophthalmologist in glaucoma. As he cannot assume the responsibility of carrying out the treatment of the disease by himself, because of the seriousness of the situation, he needs to be conversant with only two things: 1, he must not confound glaucoma with other diseases; 2, he must know how far he can go in the conservative treatment, and when to advise an operation. Positively it is not right to believe that the simplest way to deal with these questions is to send all eye cases to the specialist, for he cannot always keep such patients under his own observation and he needs aid in watching cases of chronic glaucoma.

We will deal first with the proposition that the physician should not confound glaucoma with other diseases. The two diseases with which glaucoma is constantly being confounded are

Senile Cataract and Iritis

This patient has been put off for months with the diagnosis of a senile cataract, not yet ripe for operation. Now he is incurably blind. To err is certainly human, but it must not be forgotten what it signifies when the father of a family, who should still be able to maintain them, is rendered blind by an incorrect, or a delayed diagnosis, and becomes a burden on his relatives. The confusion with senile cataract happened because the methods of examination at our disposal for the diagnosis of senile cataract were not used. Patients with glaucoma are elderly, as a rule. When the pupil is dilated in consequence of the glaucoma its area appears by ordinary daylight to be gray, because of the stronger reflex from the lens, but it has been known for a long time that this is no evidence of cataract.

Look at this elderly patient with perfectly normal eyes, whose pupils have been dilated. The gray reflex from the pupil can be seen, but, if the slight trouble is taken to investigate the eye with a magnifying glass, no real cataractous opacities can be found, and if the ophthalmoscope is used the lens is seen to be perfectly transparent. Cataractous opacities stand out in dark relief against the red background of the pupil. Whoever has impressed upon him the fact that he must use these two methods of examination before he makes a diagnosis of senile cataract will not mislead his patient by a wrong diagnosis from the mere appearance of a gray pupil.

There are other diagnostic relations between senile cataract and glaucoma which frequently demand a more careful examination. Very often both develop independently in an eye. As both diseases appear in the later years of life it is comprehensible that glaucoma may develop in an eye containing an incipient cataract, and, inversely, that cataract may appear in an eye affected with chronic glaucoma. The latter condition is called cataracta in oculo glaucomatosa.

Cataract may also be a direct consequence of the glaucoma; it is then called cataracta glaucomatosa. The eye suffering from glaucoma undergoes an increasing degeneration, the change of fluid is considerably disturbed, and the nutrition of the lens must suffer thereby. Thus it happens that we meet with opacities of the lens in eyes blinded by glaucoma. It shows itself usually by a marked swelling, a bluish white color, and a silky lustre. Extraction from such blind eyes is not only useless, but usually hazardous on account of the danger of hemorrhage.

Just as the lens may become opaque as the result of the glaucoma, so a commencing senile cataract, especially when the development and swelling is rapid, may, inversely, be the cause of a glaucomatous increase of tension with all its characteristic symptoms. We must act quite differently in such a case. We must test accurately the vision and projection, compare them with the objective condition in the lens and, when possible, in the optic nerve, and then perform iridectomy and extraction as quickly as possible.

The second disease for which glaucoma is frequently mistaken is iritis. When the first glance at an eye gives the impression that a severe inflammation is present, my advice is that in the examination of elderly persons we should not act on the preconceived idea that a certain inflammation must be present when we see a ciliary and a conjunctival injection, for this is the reason glaucoma is so often mistaken for iritis. If the facts are firmly fixed in the mind that the pupil is contracted in iritis and dilated in glaucoma the danger of mistaking these diseases for each other will be avoided. This danger is a very serious one, for cases of chronic glaucoma have been mistaken for iritis and unhappily treated with atropin. Every dilatation of the pupil in glaucoma occludes the sinus of the anterior chamber still more and makes matters worse. We do not dare to instill atropin in healthy old eyes, because an attack of glaucoma may thereby be excited. In iritis we also find obscuration of the details of the iris and the formation of exudates. In inflammatory glaucoma we meet with shallowness of the anterior chamber and cloudiness of the cornea.

The differentiation from glaucoma is more difficult than this in only one form of iritis, iritis serosa, for this disease runs its course with considerable fluctuations of the intraocular tension and sometimes the tension is high. Occasionally the pupil shows no tendency to contract, as in other forms of iritis, but may rather be of medium size without atropin. In these cases we must pay special attention to the condition of the anterior chamber; in glaucoma it is shallow, in serous iritis it is deep. The presence of deposits on *Descemet's* membrane and the history will enable a positive diagnosis to be made. An onset of iritis in gout sometimes simulates an attack of glaucoma. I do not propose that my students shall make such an error as to mistake glaucoma for iritis, and such an error will not be made if students will remember that they are studying, not to pass an examination, but for their life work, if they will not simply glance transiently at the patients I present, but study them.

Although glaucoma is not readily mistaken for cataract or iritis by the expert, yet, on the other hand, it is not always easy for the ophthalmologist himself to recognize glaucoma as such at the first glance. It is not easy to recognize a commencing glaucoma with intermittent fluctuations of tension, when the patient presents himself in the interval between attacks in the beginning of the disease. When we find the tension not increased, no excavation formed, the vision still normal, or reduced so slightly that the loss may be explained by the age alone, there is nothing to be done except to induce the patient to return when he has another attack. A suspicious tendency of the pupil to dilate frequently permits a probable diagnosis to be made.

Acute glaucoma is so characteristic that it can scarcely be mistaken for anything else, but it must be borne in mind that rare cases are met with in which the pupils are not so widely dilated.

The question whether a tumor may not be present should always be raised. If the refractive media are transparent the detachment of the retina caused by the tumor can be found as soon as it causes an increase of tension. Very rare cases have been described in which an increase of tension has accompanied a simple detachment of the retina, but, as a rule, the intraocular tension is diminished in such cases. If the refractive media are so opaque that an ophthalmoscopic examination cannot be made we have two supporting factors to consider. First, we must obtain an accurate history. If the acute condition of glaucoma has come on suddenly and the visual field is not contracted, the presence of a tumor is excluded; but if the statements of the patient indicate that a darkening of a portion of the visual field was present before the onset of the glaucoma there are several things that may be present: partial embolism of the central artery, intraocular hemorrhage, and detachment of the retina, with or without a tumor. If the lens is opaque, no explanation of the fundamental disease can be obtained by an iridectomy. In all such cases we must transilluminate the sclera with *Sachs'* lamp, in the manner demonstrated in the case of sarcoma of the chorioid.

The diagnosis of chronic inflammatory glaucoma may be difficult only when the presence of a ripe cataract renders an ophthalmoscopic examination impossible. Then we take note of the great distention of the anterior ciliary veins, the dilated pupils, the shallow anterior chamber and the increased tension. The test of the light perception and of the visual field is decisive. When we test with a light in a case of uncomplicated cataract the visual field is found to be unimpaired; it is only when such a cataract has existed many years, and then rarely, that the projection to the inner side may be not quite unimpaired, but the ophthalmologist hardly needs to take this exception into account in the differential diagnosis, because the external evidences are so characteristic that a chronic inflammatory glaucoma can be recognized. Amaurosis is also present, as a rule, when a glaucomatous cataract has lasted long, and it scarcely needs to be said that the light from the smallest lamp is perceived in uncomplicated cataract.

When a differentiation between glaucoma simplex and amblyopia due to atrophy of the optic nerve is called for, the diagnosis may be much more difficult. In doubtful cases of this nature we investigate first whether fluctuations of tension, neuralgias and obscurations have formerly occurred. Regular observations of the tension with the tonometer will show whether fluctuations of tension are present, but there are cases of glaucoma simplex in which such fluctuations either do not occur at all, or are so slight that they are within physiological limits. Then we study the excavation carefully and try to determine whether it is due to pressure, whether the vessels bend sharply at the margin and the papilla is at the same time discolored, or is atrophic. The position of the defect in the visual field may also be utilized; in glaucoma there is usually a free sector to the temporal side, in atrophy the contraction is ordinarily concentric. Finally the test of the color sense and the light sense may be employed; it is characteristic of atrophy that color perception, especially for green, is lost very early, while in glaucoma the normal arrangement of the color perceptions still exists in the little remnant of the field. Disturbances of the light sense are accustomed to appear earlier in glaucoma. This patient with glaucoma absolutum neglectum is unfortunately beyond our help and I hope that this example may prove an efficient warning.

122. Iridectomy for Glaucoma, Its Consequences and Complications

The duty of the general practitioner in the treatment of glaucoma has been pointed out, and I have indicated how far he can go with the medicamental treatment of this disease, but when such treatment is inefficient, or is not suitable, as in grave attacks of acute inflammatory glaucoma, his patients should be advised to have an operation performed. This operation should be done only by a specialist, for the operative treatment of glaucoma is one of the most difficult and responsible duties of the ophthalmologist. Still, the general practitioner should be acquainted with this part of our work, for he must not be too late in advising an operation, he must not let the right time slip by.

At the present time we have at our disposal for the operative treatment of glaucoma: 1, iridectomy; 2, sclerotomy; 3, cyclodialysis. Several other operations have been recommended, such as myotomia intraocularis, which consists of a section of the ciliary muscle, and paracentesis, either with or without incarceration of the iris in the wound. The latter operation is based on the supposition that a cystoid cicatrix is essential for the regulation of the tension of the eye for easy filtration. *Coccius* leaves the ciliary margin of the iris in the wound after a partial iridectomy, and recently *Holtz* has recommended to unite the iris with the subconjunctival tissue, an operation that has received the name of iridencleisis antiglaucomatosa.

Another operation, which has been recommended by *Abadie*, is the resection of the superior cervical ganglion of the sympathetic. This is based on the theory that a stimulation of the sympathetic induces a hypersecretion and may in this way be the cause of the glaucomatous increase of tension. It is supported by the clinical fact that paresis of the sympathetic causes a contraction of the pupil and hypotony. I do not perform this operation because it is far surpassed in its effect in acute glaucoma by the classical iridectomy, because it is still more uncertain in its effect in chronic glaucoma than the other methods, and, thirdly, because it is much too dangerous in proportion to its slight value. No one needs to die from an operation for glaucoma, but deaths have been reported after the resection of the sympathetic, and, when it is not fatal, resection of the superior cervical ganglion is frequently productive of other troubles, such as paræsthesias, dysphagia, paralyses of the vocal cords, and trophic disturbances. Such an operation can find no general acceptance.

The extirpation of the ciliary ganglion must also be considered too radical. *Kroenlein's* temporary resection of the wall of the orbit is needed for its performance. Finally it may be mentioned that *Badal* recommended the stretching and evulsion of the infratrochlear nerve, in order to alleviate the pain and reduce the tension. There is no strong indication for this operation, in my opinion; at any rate we attain the same purpose more easily in other ways. Iridectomy, sclerotomy and cyclodialysis are the principal operations for glaucoma.

I perform iridectomy in every case of acute glaucoma and in chronic inflammatory glaucoma when meiotics have failed. Cyclodialysis or sclerotomy serve as auxiliary operations in the latter class of cases. I operate in glaucoma simplex only when hypertony is demonstrable, and then prefer anterior sclerotomy. We must be clear in each individual case concerning three points: 1, we must know and be masters of the technique of the operation; 2, we must know what *we* can attain with the chosen operation in the given case; 3, we must know what *the patient* has to expect from the operation for the cure of his disease.

Technique of iridectomy for glaucoma. I consider it objectionable to operate on a patient in a sitting posture, as every muscular tension should be avoided, so I prefer to have him lie on an operating table. Local anæsthesia is secured by repeated instillations of cocain, which at the same time reduces the tension a little in many cases, but it is not unusual for general anæsthesia to be needed, when a patient has lost his self control and cannot be expected to lie quietly.

Before and during the cocainization we instill eserin or pilocarpin in order to make and keep the pupil as small as possible, for the operation is performed much more easily when the pupil is small, as the anterior chamber is deeper and we can seize the iris to better advantage. We also instill eserin in the other eye which is not to be operated on, because it has been observed repeatedly that it is apt to be attacked suddenly after the operation, as the result of the excitement. The predisposition to glaucoma is certainly present in this eye and it is prudent to prevent such an occurrence.

We wish to dissolve the connections between the periphery of the iris and the cornea in the sinus of the anterior chamber by the iridectomy, that the filtration angle may be opened, but this can be done only when the incision is placed so peripherally that we can excise a sufficient piece of the iris down to the apices of the ciliary body, hence the keratome is introduced about 1 mm from the transparent cornea in the scleral limbus, and passed forward as far as possible into the anterior chamber parallel to the surface of the iris, so that the length of the incision is about 6 or 8 mm.

When the anterior chamber is so shallow that the keratome cannot be introduced far enough we must try to lengthen the incision by moving the keratome laterally. Many surgeons use scissors for this purpose. The formation of a conjunctival flap is to be avoided because, in the first place, the vessels there are apt to bleed considerably, and in the second, a conjunctival flap favors the formation of a cystoid cicatrix. After the section has been completed the keratome is depressed somewhat and withdrawn slowly from the eye in such a way that its point is directed toward the cornea, so as not to injure the capsule and the lens as they are pushed forward. The aqueous is allowed to escape slowly at the same time, so that the tension is not reduced suddenly. In spite of all precautions it sometimes happens that the iris is partly extruded from the wound with the withdrawal of the knife. We then simply seize it with forceps and abscise it. If the iris remains in its normal position we introduce the iris forceps, open them widely, seize the iris, draw it somewhat toward the pupil in order to free the filtration angle as much as possible, then draw it out of the wound and abscise it with two strokes of the scissors.

This operation is often rendered difficult by the fact that the atrophic and narrow iris tears, or does not yield to the pulling.

The coloboma produced should be as broad as possible and extend to the margin of the iris. The corners of the sphincter must be brought back to the area of the pupil. If a tag of iris is caught in the angle of the wound we try to seize it with forceps and to abscise it, or we may introduce a small spatula and push the iris back into its normal position. Such incarcerations of the iris are dangerous, because they delay the healing of the wound, or render the regulation of the tension difficult.

After the operation has been completed it is well to determine by careful palpation the change produced in the tension. It is rightly to be expected after such an opening of the capsule of the globe, through which the aqueous has escaped, that the tension should be reduced immediately and that the eye should feel soft. This happens in many cases, but it is characteristic of glaucoma that the eyeball frequently remains remarkably tense immediately after the iridectomy.

Clinical experience has taught us that we can obtain in this way an indication of the curative effect to be expected. If the eyeball has become soft we have less anxiety in regard to the outcome, but if it remains hard we must expect, at least, that the anterior chamber will be restored more slowly than it should be. The iridectomy may be completely ineffective, the glaucoma may persist and go on to amaurosis, or the anterior chamber may reopen periodically and require weeks for its final reformation.

After we have thus investigated the immediate effect of an iridectomy for glaucoma the after treatment of the eye that has been operated on begins.

An iridectomy for glaucoma is made upward. We depart from this rule unwillingly, and perform the operation downward, or to one side, only when it has had to be repeated, as is sometimes necessary, or when the upper part of the iris is very atrophic. The reasons why the iridectomy is made upward are pre-eminently optical; cosmetic reasons are not to be taken into account when the problem is that of preserving the organ, and this is an operation to save the life of the eye, but we wish the broad coloboma, that will give rise to dazzling from the irregular refraction of the peripheral rays of light, to be covered as much as possible by the upper lid. The wound lies beneath the lid when the eye is closed, we can apply no dressing to it, but must simply wait for it to heal. Healing takes place almost invariably without infection, because the margins of the wound are quite smooth and not contused, but, to remind the patient to keep his eyes as still as possible, we bandage both of them for one day, and the eye operated on for several days more. The bandage is applied quite loosely, or simply a capsule is fastened over the eye. I consider it injudicious to apply a pressure bandage over an eye that has been operated on for glaucoma, as recommended by *Schmidt-Rimpler*. The tension reducing effect of a firmly secured pressure bandage on an unwounded eye cannot be produced on an eye that has been operated on. Such pressure must cause the wound to gape, for we cannot concentrate it upon the region involved.

The internal pressure sometimes causes the scar following the iridectomy to be uncommonly broad, and vascular soft tissue, portions of the iris, or of *Bowman's* and *Descemet's* membranes, to become interposed between the margins of the wound, while a smooth and firm cicatrization takes place after other iridectomies.

The time required for the restoration of the anterior chamber after an iridectomy for glaucoma varies a great deal and depends on the nature of the individual case. A very slow or imperfect restoration is a bad prognostic sign. Cases have been reported, as by *Mauthner* and *Schweigger*, in which the anterior chamber was not restored for years, and yet vision was saved, but they are rare exceptions.

What do we accomplish by means of an iridectomy for glaucoma? In what does its effect consist?

The great accomplishment of *Albrecht v. Graefe* consisted of the discovery that iridectomy reduces the intraocular tension in the majority of attacks of glaucoma. An iridectomy on an eye that is not glaucomatous, for example, an optical, or a preliminary iridectomy, causes no reduction of tension. How is it then that iridectomy reduces tension if glaucoma is present, but does not influence it otherwise? Various theories have been advanced, but none give a satisfactory explanation of this phenomenon.

It is generally acknowledged that the iridectomy acts primarily through the freeing of the sinus of the anterior chamber. If the periphery of the iris occupies this sinus before the operation, as is the rule in many forms of glaucoma, the peripheral excision enables the filtration of the aqueous to be reestablished. In addition to this, when the squeezing of the swollen ciliary processes against the iris ceases in the region of the coloboma the escape of the engorged blood from the ciliary processes is facilitated. Probably the secretion of intraocular fluid by the overburdened secretory organs of the eye is diminished at the same time. The evacuation of the aqueous cannot be without influence on the circulation of such an eye. The newly formed aqueous may assist in separating from the cornea the peripheral part of the iris lying upon it if the filtration angle reopens, provided that firm adhesions have not been formed, but if firm adhesions of the root of the iris are already present in the sinus of the anterior chamber, as is often the case in chronic inflammatory glaucoma, we cannot reopen the sinus entirely by the iridectomy, although the operation may still cause some improvement. We must suppose that the result is to be credited principally to the division of the adhesions. The cicatrix following an iridectomy for glaucoma is much broader than that which follows an iridectomy performed for other reasons. *Schlemm's* canal may be laid open and the outflow of the aqueous thus reestablished, especially when no atrophy of this important outlet has taken place.

The sole or preeminent cause of the curative effect of the iridectomy for glaucoma has been placed by many in the nature of the scar in the sclera. It has been believed that a "filtration cicatrix" was formed, through which the intraocular fluids could pass. Others again believe that the capsule of the globe is relaxed by the scleral cicatrix. This is of direct benefit to the cases in which the outer layers of the sclera are too rigid, the capsule of the globe, as it were, too small.

A final group of ophthalmologists look upon the exsection of the iris as the most important feature of the operation. Those who refer glaucoma to nerve irritation believe that the iridectomy acts through the section of the nerves. Others suppose that a direct communication is formed between the vitreous and the anterior chamber by the excision of the iris, so that the vitreous can transudate into the aqueous. Finally, it has been thought by *Exner* that a decrease in the vascular tension in the eye was obtained by the excision of the iris, in which the smaller arteries, veins and capillaries are removed, leaving only the stumps of the larger arteries and veins at the ciliary margin, so that, as anastomoses form between these, the arterial blood flows directly into the veins, relieving the pressure in the arteries, and consequently the intraocular tension is reduced. It is hard to find a uniform explanation of the tension reducing effect of iridectomy. Fortunately for our patients, this is not necessary; it is much more important that the iridectomy is of service to the patient.

What has the patient to expect from an iridectomy for glaucoma as regards its influence on his disease?

The immediate effect of iridectomy in acute glaucoma is, as a rule, the cessation of the frightful pain, either instantly or in the course of a day or two. At the same time the signs of engorgement retrogress, the great injection disappears, and the refractive media become clear, so that the fundus can be seen, after a few days. The patient soon rejoices in an improvement of his vision, but usually several weeks elapse before the best vision is obtained, which the eye generally keeps. This is due partly to the astigmatism caused by the wound, partly to changes in the retina.

The final result of iridectomy in an attack of acute glaucoma is generally good, sometimes it is brilliant. The vision may regain the normal after a first attack. When such an eye is examined the tension is found to be normal, the cornea to be clear and to have its sensibility reestablished. Permanent lesions remain only in the pupil and iris. The pupil remains moderately dilated and no longer reacts promptly. The greater the tendency of the pupil to dilate, the more doubtful is the final result. Gray places can frequently be seen in the iris, which are due to atrophic thinness. The eye may be preserved with good vision for many years. Regular reexaminations are necessary, and we usually need the further support given by meiotics. When several attacks of acute glaucoma have occurred, or a contraction of the visual field and an excavation of the papilla existed before the last attack, the prognosis is bad.

In chronic inflammatory glaucoma, when the result is good, it takes a longer time for the maximum of vision to be attained; the anterior chamber becomes deeper, but such eyes are accustomed to retain the habitus glaucomatosus. Two symptoms are interesting in these cases. *Graefe* first noticed that even a distinct pressure excavation may become more nearly level and so be transformed into a shallower cup. A whitish discoloration of the papilla may be so marked that one is startled at the first glance and is led to expect to find the vision greatly impaired, but the latter is not always the case. *Graefe* thought that the relative positions of the nerve fibres were changed by this alteration in the excavation so that the atrophic fibres became more clearly visible.

The prognosis naturally is bad in chronic inflammatory glaucoma when the retina and optic nerve have already been seriously injured; therefore I have called attention to the fact that we must not delay an operation too long. Unfortunately operative intervention cannot prevent the further loss of vision in a great many cases, and because of this dilemma I always try the conservative treatment first in this form of the disease. The best that a patient with chronic inflammatory glaucoma can hope for is that the disease may be delayed as long as possible in its development.

Certain complications may arise even when the iridectomy is followed at first by a favorable course. Like most operations on the human organism this one may result in certain disadvantages, a knowledge of which is necessary to the right appreciation of our therapeutic ability.

First among these drawbacks and complications are intraocular hemorrhages. When the increased tension in an eye is reduced very suddenly the walls of the blood vessels may become more permeable; this is the reason why we meet with these hemorrhages chiefly after iridectomy in acute and chronic inflammatory glaucoma. We find quite regularly little hemorrhages in the retina after this operation for acute glaucoma, situated usually in the vicinity of the papilla and macula, but sometimes in the region of the equator. Ordinarily they are of no serious importance and are absorbed in the course of six or eight weeks. When the tension was not very high before the operation the retinal hemorrhages are not so frequent, nor so regular; they are scarcely to be met with in noninflammatory glaucoma, even though the tension was increased at the time of operation. This indicates that the reduction of tension is not their sole cause, and we must suppose that the walls of the vessels themselves are more seriously injured in inflammatory than in noninflammatory glaucoma.

Hemorrhage into the anterior chamber occasionally forms an unpleasant complication, although it is not as common, nor as great, as after an iridectomy for chronic iritis. It can hinder the return of the tension to normal for days through the transient obstruction of the filtration angle.

Rupture of retinal hemorrhages into the vitreous after iridectomy is more rare. Only a few cases have been reported in which such a severe, destructive hemorrhage occurred as is occasionally seen after an extraction of cataract.

Another very unpleasant drawback to the iridectomy for glaucoma is the appearance or the rapid development of an already existing cataract. Various conditions unite to bring this about. The contact of an instrument with the capsule of the lens, and slight injuries of the latter, that are by no means immediately visible, cannot always be avoided when it is introduced into the shallow, or totally abolished anterior chamber, even with the most brilliant technique. It is also probable that, with no fault on the part of the surgeon, little lacerations may be produced sometimes in the capsule by the rapid diminution of tension and the swift advance of the lens, as has been shown by the histological findings of *Sachs*. An already existing cataract has been repeatedly observed to progress after an iridectomy. I have demonstrated experimentally that the diminution of tension leads to a temporary change in the composition of the intraocular fluid, which may be permanent in eyes with diseased vessels. A more rapid development of a cataract may therefore be induced in an individual whose fluids are not suited for the nutrition of the lens. Usually these lenticular opacities appear some days or weeks after the iridectomy, and then the extraction of the lens must be added.

Another condition occasionally met with after iridectomy for glaucoma is the so called cystoid cicatrix, to which attention was called in the study of the healing of the wound after an operation for cataract. Ordinarily a smooth, white, delicate scar, which is scarcely visible, remains after an iridectomy, but occasionally the margins of the wound separate a little after a few weeks. The glassy looking connective tissue that forms the scar gives way before the intraocular pressure, and a single, vesicular protrusion develops, which is covered by conjunctiva. Opinions differ as to the frequency of this cystoid cicatrix. Some think that the scar becomes somewhat broader in all eyes in which the tension is increased, even though no protrusion takes place. According to *Graefe* a cystoid cicatrix is formed in about one fifth of the eyes operated on for glaucoma. This kind of cicatrix is most likely to appear when the iris is caught in the wound, and as this usually happens in the angles it is here that the cystoid cicatrix is most apt to develop.

Not a small number of ophthalmologists are convinced that a broad, transparent and cystoid cicatrix after an iridectomy favors the cure of the glaucoma. Some operative procedures have been devised for the purpose of obtaining such thin cicatrices through which these surgeons think that the aqueous may filter. We do in fact occasionally observe that the aqueous breaks through the tissue of the cystoid cicatrix and is effused beneath the conjunctiva; hence we need not interfere, ordinarily, with a cystoid cicatrization after an iridectomy for glaucoma, and yet we do not discharge such patients without some anxiety, for they should be kept under observation. When the cystoid cicatrix has attained large dimensions it provokes conjunctival irritation and we have to do away with the ectasia by carefully cauterizing it repeatedly, so as to flatten the scar, or by opening and excising the cyst, closing the wound, preferably, by a conjunctival plastic operation. Such eyes are threatened by another danger, the same that threatens eyes in which cystoid degeneration of the wound has followed an operation for cataract: the thin covering may rupture easily and a rapidly advancing infection may take place. When there is the least suspicion of the presence of such an infection, which may happen years later, the patient must return to a specialist for cauterizations and a plastic operation.

It has already been mentioned that after an iridectomy on the first eye an unexpected attack of glaucoma may take place in the second.

The vision deteriorates rapidly after an iridectomy in a small percentage of cases. In these we see the papillæ become paler, the vessels smaller, the excavations deeper, the visual fields more and more contracted. Hence in the brilliant operative treatment of glaucoma we can only hope for the best for our patients, we can promise nothing, and must be satisfied when we have helped them for a time.

123. Cyclodialysis and Sclerotomy for Glaucoma

Our first patient has chronic inflammatory glaucoma. The anterior ciliary veins about the cornea are distended, the cornea itself is not quite clear, the anterior chamber is very shallow, the pupil is widely dilated, the iris is reduced to a narrow band, the tension is considerably plus. It is imperative that the iridectomy be carried absolutely to the periphery to save such an eye, but this would be very difficult to do with such an extremely shallow anterior chamber, so I shall first perform cyclodialysis.

This operation was devised by *Heine* and was based on the fact, to which I called attention while speaking of the after treatment of the operation for cataract, that after extraction with iridectomy, the anterior chamber may become shallow without a reopening of the wound, some days after the latter has healed faultlessly. Such an eye is soft, and *Fuchs* has demonstrated that a temporary detachment of the chorioid has taken place. *Fuchs* supposes that the pectinate ligament is loosened by the iridectomy so as to form a communication between the anterior chamber and the suprachorioidal space. *Axenfeld* conjectured that possibly the curative action of iridectomy in glaucoma could be explained in this way, in that the aqueous was resorbed from the suprachorioidal space. *Heine* utilized these suggestions and tried to establish such a communication between the anterior chamber and the suprachorioidal space in the following way:

After the eye has been cocainized I make an incision through the conjunctiva, that has been lifted up with forceps, about 8 mm from the limbus, below and to the outer side of the cornea in this case, push aside the episcleral tissue and separate the margins of the wound so as to lay bare the sclera. Now I seize the conjunctiva with fixation forceps close to the diametrically opposite margin of the cornea, in this case above and to the inner side, so as to hold the eye firmly. Next I make an incision in the bared sclera, 5 or 6 mm from the limbus, parallel to a tangent to the corneoscleral margin at this point, and gradually divide the fibres until the sclera has been cut through. *Heine* was right when he said that we can instantly tell by the feeling when the sclera has been perforated. I widen the incision until it is 2 or 3 mm long. Bleeding sometimes occurs and careful sponging is necessary. Then I introduce a stylet into the wound and pass it along the inner surface of the sclera. This is, in my judgment, the most important feature of the operation. I must be certain that the stylet is applied to the inner surface of the sclera and does not injure the portion of the ciliary body that is in front of it. I press the instrument firmly against the sclera and pass it between this membrane and the ciliary body to the limbus, until I feel the resistance of the ligamentum pectinatum, another important moment; I press it slowly forward, the resistance is overcome and the instrument appears in the anterior chamber. I have succeeded in passing it between the cornea and the closely approximated iris, the ciliary body is detached and there is a communication with the anterior chamber. Now I make lateral excursions with the instrument, so as to detach the insertion of the iris over a larger area and enlarge the cyclodialysis, and then withdraw it from the wound. There is no escape of aqueous. The wound in the conjunctiva is closed with a suture and the operation is finished. A dressing is applied and the patient put to bed.

What do we expect to gain from this operation? I am convinced, as are other ophthalmologists, that cyclodialysis is able to diminish the increased intraocular tension in certain cases of glaucoma, and in some cases it has resulted in a marked and permanent hypotony. If the tension is reduced in the eye of this patient, which will be known tomorrow, so much the better, we will stop right there. If the cyclodialysis should not produce this effect we will at least hope for something else. As a rule, the shallow anterior chamber becomes deeper a few days after the operation, and then, if the tension is not satisfactory, an iridectomy can be performed under conditions more conducive to success than they were today.

Cyclodialysis is still a very young operation, the questions concerning it have not yet been settled and the discussion as to its value displays the customary literary strife among ophthalmologists. It has its enemies and its friends. *Schnabel* condemned it strongly without having performed it even once. *Krauss* raises the obvious consideration, from the results of his experiments on animals, that the operation cannot create a permanent communication between the anterior chamber and the suprachorioidal space, because a readhesion takes place. Partial lesions of the ciliary body and of the iris have been found at the site of the operation in some of the eyes that have later been examined pathologically. None of these objections can get rid of the clinical fact that cyclodialysis can reduce the tension in a number of cases of glaucoma of the human eye for a considerable period, and therefore is of good service in the same way as iridectomy. The statement that iridectomy does not do away with glaucoma as a disease, but only eliminates the mechanical factor of increased tension, is equally true of cyclodialysis; therefore we can make use of one operation just is well as of the other. If anatomical researches reveal a reattachment of the detached portion, and furnish no explanation of the tension reducing effect of cyclodialysis, the same is true of iridectomy, the real nature of the action of which in glaucoma is still unknown, in spite of all theories and researches.

On the other hand there is no doubt that too exacting demands have been made upon the services of cyclodialysis by its opponents, as frequently happens when a new treatment is tested. Cyclodialysis cannot restore the functions of an amaurotic eye. We must not forget that the operation was tried at first in the worst cases of glaucoma, in which there was not much to be lost. In such cases it is difficult, if not impossible, to comply with the requirements laid down by *Krauss*, that 1, the tension shall be permanently diminished; 2, the signs of irritation shall permanently disappear; 3, the excavation of the papilla shall not advance; 4, the refractive media shall become clear; 5, the function shall be restored; 6, the painfulness shall pass away; 7, complications shall be avoided.

The friends of the operation should not speak of permanent effects, as they have spoken. When the results of iridectomy are not accepted as permanent until after the lapse of decades, those of cyclodialysis should not be said to be permanent when the first few cases have been under observation only about two years. The history of the treatment of glaucoma should be taken rather more into account. My own opinion, drawn from my clinical observations up to the present time, is that cyclodialysis cannot, will not, and need not universally take the place of *v. Graefe's* iridectomy for glaucoma. Until its definite place in the treatment can be determined, which will not be possible for a long time to come, I shall consider it an auxiliary operation. As such it has certain advantages over iridectomy in quite a number of cases.

The operation is one that is easily performed, preserves a round pupil in the eye and is much less dangerous than an iridectomy when we are obliged to operate at a time when the anterior chamber is totally abolished, when the tension is high, when hemorrhage is to be feared, when the iris is atrophic, and when an iridectomy has already been performed. Under such circumstances it is sometimes the only operation that can be performed, it can be repeated more readily than any other, and it does not preclude any other subsequent intervention. Whether these advantages will suffice to give it a permanent place in the operative treatment of glaucoma must be determined by long clinical observation. On the whole I am a friend to the conservative treatment of glaucoma, but when I have decided that it is best to operate I take cyclodialysis into consideration. I employ it chiefly in cases in which it would be difficult to perform an exact iridectomy, mainly in cases of chronic inflammatory glaucoma with shallow anterior chamber and a narrow iris. I also use it in cases of glaucoma simplex in which fluctuations of tension are demonstrable. In acute glaucoma, on the contrary, I perform iridectomy, because the effect of cyclodialysis is not produced with sufficient rapidity. Iridectomy is likewise the first recourse in secondary glaucoma from seclusio pupillæ, or leucoma adhærens.

A third operation for glaucoma, sclerotomy, introduced by *Quaglino* and perfected by *de Wecker*, is performed in many ways and with various modifications, which may be divided into two groups: a sclerotomy performed in the limbus is called sclerotomia anterior, one performed farther back in the sclera, through which a portion of the vitreous is removed, is known as sclerotomia posterior.

Anterior sclerotomy emanated from the idea that the essential point in an iridectomy for glaucoma is the creation of a broad scar, permeable to the aqueous, a "filtration cicatrix," and that therefore the iridectomy could be superseded by sclerotomy. The effort is also made to open *Fontana's* space as freely as possible by the incision. Permanently good results can be obtained by this operation in a number of forms of glaucoma. In glaucoma simplex especially sclerotomy surpasses iridectomy, and is followed much less often by a direct loss of vision. I therefore prefer sclerotomy when any operation is necessary in glaucoma simplex, when it has been determined that the process in the two eyes cannot be arrested by meiotics. I introduce a *Graefe* knife in the margin of the cornea, make a counter puncture on the other side, and cut upward as if I were about to make a narrow flap for a cataract extraction. As soon as two thirds of the incision have been completed I open the wound, so that the aqueous will escape slowly. This must be done very carefully. The iris will arch over the back of the knife, but will not prolapse. When the aqueous has been evacuated I withdraw the knife slowly, leaving a portion of the sclera uncut in the middle of the wound. The after treatment is the same as that of other operations for glaucoma, a bandage and regular instillations of eserin. Many surgeons endeavor to combine an iridodialysis with this sclerotomy.

124. The Change of Fluid in the Normal Eye

If there is a disease of the eye that is governed by a disturbance of the intraocular change of fluid, that disease is glaucoma. This is evident not only from the clinical picture but also from the pathology of the disease. Generations of investigators have labored, and must continue to labor, over the subject of the change of fluids in the eye. *Leber* especially has won great credit in this field. The extent of, and the difficulties in this field of research preclude that I should enter into details in the short time at our disposal. The subject is studied in the clinic almost entirely to facilitate the understanding of the glaucomatous process and for this purpose I will give a brief review of the theory of the change of intraocular fluid and at the same time demonstrate some experiments.

The intraocular fluid, which fills the anterior chamber, the posterior chamber and the vitreous, has two physiological duties to perform. It is a nutritive fluid and as such must conduct nutritive material to the transparent, nonvascular organs of the eye, the lens, the vitreous and the cornea, and must remove the products of metabolism at the same time. It must also constantly maintain the form and curvature of the walls of the eye. We became acquainted with the latter function in the study of phthisis bulbi. When the tissue of the vitreous is lost, when the intraocular fluid is no longer formed in sufficient quantity and of normal composition, the capsule of the eyeball may still be well nourished, but the eye atrophies.

I. Does a Change of Fluid Take Place in the Eye?

Is there a constant renewal of the intraocular fluid? Everywhere else the blood and lymph are in constant motion, this is a change of fluid. The cells of the organ draw their nutriment from the lymph and blood capillaries, by the molecular powers of diffusion and osmosis, as well as by specific, chemical affinities, and exchange for it their catabolic products. This is metabolism. The two processes work hand in hand in the organism, but we must not mistake the one for the other. Metabolism takes place when an organ is nourished, it takes place within the eye, but does a change of fluid likewise take place there?

We learned in our studies concerning the processes of nutrition in the lens and cornea that these nonvascular organs depend for their nutrition on osmosis, diffusion and the specific biological affinities of the cell protoplasm. Does a change take place in the surrounding fluid in the physical sense of a current? Or does the intraocular fluid form an exception to the rules of the movement of the blood and lymph that prevail in other organs? This is the first question to be answered. Three theories have been advanced.

1. Theory of a Rapid Change of Fluid.

Ophthalmologists learned long ago from their operations that the anterior chamber refills very quickly after the escape of the aqueous. If the aqueous is removed from the eye of a rabbit with a syringe, it will be replaced in a few minutes, and the involuntary conclusion is that the fluid is renewed rapidly in the intact eye.

The appearance of crystalloid substances in the aqueous, soon after they have been introduced into the organism, also indicates a rapid renewal of the intraocular fluid. For example, if we inject a solution of potassium iodide into a rabbit the drug appears in the aqueous after a very short time. We may use coloring matters, like fluorescin, in a similar way, according to *Ehrlich.* These crystalloid substances are soon excreted again from the eye, especially from the aqueous, and some authors have believed that they could obtain data in regard to the rapidity of the renewal of the aqueous from the time necessary for the disappearance of these substances. How quickly such diffusible substances disappear from the aqueous and vitreous may be shown by the curves worked out by my assistant, *Dr. Loehlein,* for other pharmacodynamical reasons.

Still the conclusion cannot be drawn from all these observations and experiments that the intraocular fluid is renewed very rapidly, that there is a lively change of fluid in the eye, for the aqueous newly secreted after a puncture is not normal. It coagulates spontaneously, in contrast to normal aqueous, and contains from fifty to eighty times the normal amount of albumin. The puncture reduces the intraocular tension suddenly and this is not a normal condition for the formation of intraocular fluid.

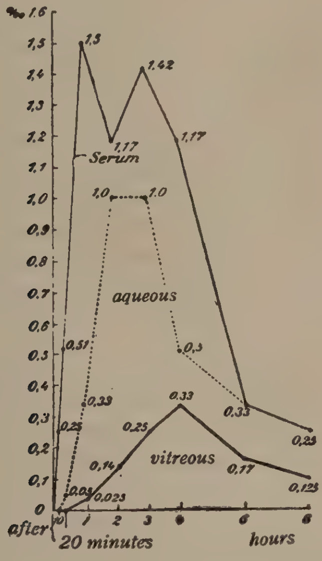

FIG. 101.—Per mille of Potassic Iodide in Serum, Aqueous, and Vitreous after Subcutaneous Injection of 0.3 ccm of a 20% solution to 100 grams Weight of Animal.

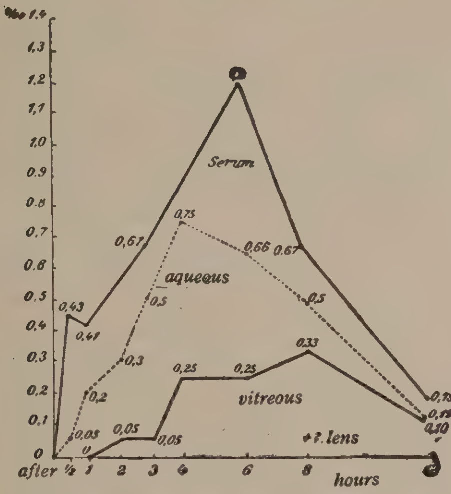

FIG. 102.—Per mille of Potassic Iodide in Serum, Aqueous, and Vitreous after Feeding an Animal a 20% solution in the Proportion of 0.3 ccm to 100 grams of Weight.

The fluorescin does not make visible a current of the intraocular fluid, because it spreads largely by diffusion, which must not be mistaken for a physical current. This is shown by the fact that, according to the researches of *Ehrenthal,* the same symptoms are produced within the eyes of dead animals in which the movement of the blood and lymph are naturally excluded, and if foreign crystalloid substances, like potassium iodide, fluorescin and potassium ferrocyanide, disappear from the intraocular fluid during life through diffusion, it is evident that we learn nothing by this experiment concerning the rapid renewal of the intraocular fluid.

2. Theory of No Movement of Fluid.

The theory that a very rapid current of intraocular fluid circulates within the eye is diametrically opposed to the theory of *Weiss*, that the presence of a movement of fluid in the eye is absolutely unproved, and that an absence of such a current is perfectly conceivable.

The overwhelming majority of ophthalmologists are unable to subscribe to such a negation. The golden mean lies between these two extremes, and at the present time we believe in a very slow current of the intraocular fluid.

3. Theory of a Slow Movement of the Fluid.

The answer to this question would be quite simple if we could render visible the movement of fluid in so transparent an organ, but this is impossible. We can detect no trace of movement, even when minute bits of gold leaf and cholesterin crystals are introduced into the anterior chamber and the eye is kept quiet. This is not surprising when we remember that, according to the calculations of *Leber*, a particle of fluid needs at least fifteen minutes to pass from the centre of the pupil to the sinus of the anterior chamber.

As we cannot see the current of intraocular fluid we have to consider what other data we possess to prove the presence of a real change of fluid. From the moment of death, when the blood pressure is removed, the eyes continually grow softer, even when protected from external evaporation, showing that, with subnormal pressure, fluid flows out of a fresh, dead eye. If we raise the tension above normal by introducing under external pressure fluid into the anterior chamber of such a fresh dead eye through a canula, as done by *Leber*, we find that fluid constantly filters out of the eye and we see it trickle from the ends of the cut ciliary veins. If fluid flows from the eye under subnormal and supernormal pressure we are justified in supposing that a change of fluid takes place when the pressure is normal.

It can be easily shown that fluid disappears by resorption from the living eye, by injecting physiological salt solution into the anterior chamber. The increase of tension thus produced soon diminishes because of this resorption. Under normal conditions the quantity of fluid secreted in the eye in a unit of time is the same as that removed by resorption or filtration. This filtration of fluid from the eye that we can establish in the freshly enucleated organ is not very different from that which takes place during life. Therefore if we are able to determine the quantity of fluid which leaves the freshly enucleated eye by way of filtration into the veins when the tension of the eye is normal, we can at least obtain an idea how much intraocular fluid passes through the eye in a certain time.

Leber constructed a special instrument, the filtration manometer, for this experiment, the principle of which is that a current of physiological salt solution is introduced into the eye under a certain fixed pressure, the quantity of which may be measured by the capillary marks on the tube.

Many experiments by *Leber*, *Priestley Smith*, *Hamburger*, and *Niesnamoff* with such instruments, which deal only with the conditions in the anterior chamber, have shown that about one hour is necessary for the secretion of the same quantity of aqueous as fills the anterior chamber. From this we may conclude that the movement of fluid in the anterior chamber, while distinctly present, is very slow.

It is evidently slower than appears in these experiments with the filtration manometer, because errors appertain to this method of examination. The fluid flows more easily into the veins which are connected with *Schlemm's* canal in dead than in living animals, as *Leber* himself pointed out. The anterior chamber may be dilated also, and the filtration through the sinus considerably facilitated *(Priestley Smith)*.

A second method by which the outflow of the intraocular fluid may be determined is given by *Leplat*. Fluid under a pressure of 25 mm of mercury in a tube is placed in communication with the cavity of the vitreous. The fluid shows no movement at first, but as soon as vaseline is injected into the anterior chamber of the living rabbit, so as to occlude the principal outlet, a certain quantity of fluid escapes from the canula. The results are analogous to those obtained with the filtration manometer. The conditions in the eye were certainly not normal, but rather those of violent interference, in these experiments, but they show at any rate that the movement of the fluid in the eye must be very slow. According to *Wessely's* calculations, from the decrease of the albumin in the aqueous after puncture, two hours must probably elapse before a sufficient quantity of intraocular fluid has been formed to fill the anterior chamber.

Aside from such experimental researches, which prove a slow change of fluid, we have theoretical reasons that render it probable.

Strong currents are contraindicated because one duty of the intraocular fluid is to maintain the form of the eyeball. In addition to this, the transparent media of the eye, the cornea, lens and vitreous, have only a slow metabolism, because they have in reality only an optical function, not a specially active one which would necessitate an accelerated movement of the fluid. On the other hand, it must be remembered that fluid flows into the eye with the least increase of the external pressure and that with the least reduction of this pressure fluid flows out. This also confirms the theory that the change of fluid in the normal eye is slow.

II. Origin of the Intraocular Fluid

It is evident that the eye contains an organ that secretes fluid, and it is just as plain that this organ can be found only in the uvea. The chorioid is excluded as the secretory organ, because if it had to secrete the principal part of the intraocular fluid the latter would be continually percolating through the retina, which a consideration of its delicate structure shows to be impossible. Yet the chorioid was formerly thought by *Knies* and *Ulrich* to be secretory.

We also know from the experiments of *Deutschmann* that after extirpation of the ciliary body and iris the entire production of intraocular fluid ceases, and that such eyes become phthisical with atrophy of the vitreous. The chorioid therefore cannot even secrete that part of the intraocular fluid which supplies the vitreous. Hence its source must be in the anterior segment of the uvea and the only question is whether the secretory organ is the ciliary body, the iris, or both combined. This question is not yet settled.

The answer does not seem to be difficult at first glance. When we look at a section of the human eye the ciliary body with its ciliary processes seems to be called upon a priori to play the part of a secretory organ, for its surface is enormously enlarged by the formation of the ciliary processes, which are richly supplied with blood vessels. By their structure they remind us of glands, particularly of those in which the secretory epithelium is situated on the outside, but the ciliary processes are not true glands, and the resemblance is superficial. The pathological reasons that indicate the secretory function of the ciliary body are more valid.

Let us examine two sections under the microscope. The first section comes from an eye with exclusion of the pupil. The posterior chamber is much distended and the impression given is that the distention has been produced by secretion from the ciliary processes. The other section, taken from a staphyloma in which the iris and cornea were adherent, likewise indicates increased activity on the part of the ciliary processes. These indications of the secretory function of the ciliary body have been known for a century (*v. Beer*).

Experimental evidence has appeared in modern times. *Deutschmann* removed by evulsion the iris and ciliary body, the consequence of which was that the production of aqueous ceased and the vitreous atrophied. The only conclusion to be drawn from this is that the iris and ciliary body together secrete the intraocular fluid, so we have to call for assistance on an observation made on man which makes it probable that this function appertains chiefly to the ciliary body. The entire iris may be torn away without injury to the normal formation of the intraocular fluid. Total absence of the iris may also be congenital. Hence, the iris alone, but not the iris and ciliary body together, may be removed from the eye and the normal fullness with intraocular fluid be maintained.

The second kind of experiment, from which it was hoped to learn the place where the intraocular fluid is formed, consists of the introduction of foreign substances, like potassium iodide, and potassium ferrocyanide, in various ways into animals and then tracing their extension into the eye by chemical reactions. These experiments, which have been numerous, could not attain the desired end because the extension of these substances is governed too much by the laws of diffusion. How little can be learned in this manner concerning the place where the intraocular fluid is secreted may be realized best, perhaps, from the fact that after injections of potassium ferrocyanide exactly the same zones of reaction can be demonstrated with ferric chloride in the dead eye as in the living. It can be proved that potassium iodide appears first in the aqueous and then in the most anterior layers of the vitreous, which it gradually permeates.

In contrast to these abortive experiments the suggestion to make observable during life the movement of fluid in the normal eye by the introduction of coloring matter has attracted much attention. It is remarkable that this scientific thought occurred to no ophthalmologist at the time when these questions were being disputed. Ophthalmology has to thank the surpassing genius of *Paul Ehrlich* for the advance furnished by the fluorescin method.

If a suitable dose of a solution of fluorescin is injected into the blood current of a rabbit it appears in a few minutes in the anterior chamber, in the form of a vertical, curved, green line on the posterior surface of the cornea, starting from the periphery of the iris. How the line is produced has not been fully explained; it is met with only in rabbits. *Ehrlich* himself believes that eddies of fluid come from both sides of the iris and come together in the median line, but we know that such currents of fluid do not exist in the eye. Besides, the law of diffusion enters so much into the excretion of the fluorescin from the body and into its appearance in the eye that the line of fluorescin does not of itself reveal the processes of secretion. The importance of the fluorescin method by no means depends on that of *Ehrlich's* line, as many have erroneously believed. A phenomenon that can be made visible in only one kind of animal, the rabbit, and is greatly dependent on the quantity of fluorescin employed in the individual experiment, evidently cannot answer the question as to the change of fluid.

Two important symptoms of the metabolism and change of fluid in the eye have been added to our knowledge by the fluorescin method. The fluorescin escapes diffusely from the vessels of the iris in all kinds of animals, not rabbits alone, and the most interesting point of this phenomenon is that this escape cannot be explained simply by diffusion, but is connected with the process of secretion. *Wessely* has shown that this escape of fluorescin from the iris is proportioned to the size of the vessels. If the eye is irritated so as to produce a hyperæmia the escape of fluorescin increases, and the reverse happens if vasoconstriction is produced artificially. If we try to explain this phenomenon by an enlargement or diminution of the surface from which diffusion takes place, caused by the change in calibre of the vessels, we find that the escape of fluorescin from the ciliary body is demonstrably parallel with the qualitative and quantitative change of the aqueous secreted at the same time. The fluorescin method gives us the first evidence of secretory processes in the iris.

The second experiment of *Ehrlich* is to puncture the anterior chamber of a rabbit, after it has received an intravenous injection of fluorescin, when the newly formed aqueous will be seen to pour through the pupil from the posterior to the anterior chamber in the form of thick, green streams. The ciliary body of such an animal is found on dissection to be distended with fluorescin. This proves that the aqueous which is newly formed after reduction of the tension by puncture comes from the ciliary vessels. We may convince ourselves of this fact anatomically. *Greeff* has shown that after punctures of the anterior chamber the epithelium of the ciliary processes may be detached in the form of bullæ, in which coagulations of albumin and fibrin take place.

We do not need such severe irritations to prove the secretory activity of the ciliary processes by the help of fluorescin; with much weaker ones we may perceive the origin of the intraocular fluid from the ciliary processes by the greater amount of albumin in the aqueous and the corresponding amount of fluorescin. Thus a gradual transition from pathological to normal conditions may be established.

We have still another experiment, for which we are indebted to *Leber*, which shows that the principal part of the secretion after puncture comes from the ciliary processes. If, after abscission of the cornea, a little tube is introduced into the pupil so as to guide the secretion of the ciliary processes outward the anterior surface of the iris remains almost dry, a sign that under such circumstances its secretion is very slight.

We have thus obtained data from the normal anatomy, the pathology and experiments that indicate the ciliary processes to be the secretory source of the intraocular fluid. Recently some investigators have claimed that the iris takes part in the process of secretion. *C. Hamburger* in particular has won credit by bringing this question up again in the face of dogma and authority. He correctly infers a participation of the surface of the iris in the secretion of aqueous from the behavior of the anterior chamber in seclusio pupillæ. The prolonged presence of fluid in the anterior chamber in such a condition cannot be understood except through the assumption of a secretory function of the anterior surface of the iris.

In embryonal life, at a time when the pupil is still occluded by the pupillary membrane, we find an anterior chamber already present; this also is ascribed by *Hamburger* to a secretory activity of the iris, but it is difficult to determine whether this is so or not, because we do not know that the pupillary membrane is quite impermeable. The traces of fœtal aqueous may perhaps come from the vessels of the pupillary membrane, and even if the iris secretes during fœtal life it does not follow that it needs to do so later. *Hamburger* also believes that in irideremia the ciliary body acts vicariously for the iris, but this has not been proved. He goes so far as to suppose that the iris alone secretes the aqueous in the normal eye, but that exceptionally, under certain conditions, the ciliary processes may furnish this secretion. He believes that the ciliary body secretes at the most only a small quantity of fluid for the vitreous, but produces aqueous only when it is irritated, or other high demands render the new formation of intraocular fluid necessary. These views *Hamburger* deduces chiefly from his theory of "the physiological exclusion of the pupils."

The theory of the physiological exclusion of the pupils assumes that under normal conditions fluid may not be able to pass through them. As the iris is arched forward by the anterior surface of the lens the force exerted by the sphincter pupillæ in its contraction may be directed partly against the lens and so exclude the pupil; this would render a continuous current from the posterior chamber impossible and, in that case, the iris alone could secrete the aqueous under normal conditions.

The basis of this theory is an experiment in which *Hamburger* injected a minute drop of fluorescin into the posterior chamber and found that this very diffusible coloring matter did not pass at once through the pupil into the anterior chamber, as would be expected if an open communication were present. More than twenty minutes often elapsed before it appeared. When the eye was irritated or punctured the fluorescin appeared immediately in the anterior chamber. This experiment is beyond doubt a very important one, which deals a bad blow to the old theory of the preponderating and sole secretion of the aqueous by the ciliary body, but its technique is so full of minutiæ that such serious errors may be made as the injection of the fluorescin into the lens, or into the vitreous, which would explain its late appearance in the anterior chamber. For this reason this experiment does not suffice, in the opinion of most ophthalmologists, to prove the theory of a physiological exclusion of the pupil. The fluorescin is held back in the posterior chamber more markedly in such experiments, according to *Wessely*, when a strong current of secretion from the ciliary body is caused to take place by means of subconjunctival injections of salt. This particular kind of diffusion still needs an explanation. I am inclined to believe that the fluorescin does not appear so quickly in the anterior chamber in *Hamburger's* experiment because resorptive forces immediately seize upon the coloring matter and try to discharge it into the circulation. The organism frees itself so quickly of this foreign substance after an intravenous injection that only a small percentage of the entire quantity is to be met with in the blood at the end of five minutes (*Loehlein*).

Some physiological facts contradict this theory. The physiological exclusion of the pupil must be firmest when the sphincter is contracted by accommodation and by the action of eserin, but we know that in accommodation the lens shakes distinctly and sinks somewhat downward, showing that the iris is not firmly adherent. It has also been ascertained that when India ink is injected into the vitreous, granules of this substance may appear in the pupil and anterior chamber, during life, at the end of half an hour. Moreover, we do not know that the tone of the sphincter pupillæ is so strong that it cannot be overcome by the secretion of the ciliary body.

Above all the lower surface of the iris is so irregular—it is necessary to take only the pigment coat into consideration—that it is doubtful if so close an exclusion is possible. These factors explain why *Hamburger* has succeeded only in convincing the majority of ophthalmologists that the iris participates in the production of the aqueous. His idea that the iris is the sole source of the aqueous, and that the ciliary body acts only as a reserve organ, is not accepted at the present time. The iris and ciliary body both take part in the formation of the intraocular fluid, but whether the iris produces the aqueous, and the ciliary body the vitreous fluid, under normal conditions, or whether the activity of the ciliary body preponderates, we do not know.

III. The Outlets for the Intraocular Fluid

The principal outlet for the aqueous is *Schlemm's* canal, which was formerly thought by anatomists to be a lymph space, but was proved by *Leber* to be a vein. It lies embedded in the firm tissue of the sclera and forms a venous ring connected laterally with the episcleral veins. Under the microscope its wall is seen to consist of a simple tube of endothelium. No wide opening exists between the anterior chamber and this canal, and yet we know with certainty that the aqueous leaves the eye through this passage. One proof of this has been demonstrated already; if this outlet is obstructed the increased tension of glaucoma appears in the eye. Another proof is gained from the results of numerous experiments with the filtration manometer. When fluid is injected under constant pressure into the anterior chamber of a fresh, dead eye, it escapes by way of the veins and trickles from the cut ends of the anterior ciliary veins. If a staining solution, like carmine, is injected into the anterior chamber, it passes readily into the sinus venosus. Injections of India ink into the living eye show that after its introduction into the anterior chamber minute granules can be found in both the sinus venosus and the episcleral veins (*Nuel, Asayama*). Therefore there is no doubt of a connection between the anterior chamber and *Schlemm's* canal, which is situated in the sinus of the anterior chamber. The way of communication is afforded by the apertures in *Fontana's* space, the clefts in *Descemet's* membrane, which is loosened in texture at this place, and the intracellular cement edges of the endothelium in *Schlemm's* canal itself.

Since the aqueous passes through such fine and minute spaces outward into this venous plexus its passage seems to be only a sort of filtration.

The vessels of the iris partake in the resorption of the aqueous and form a second outlet from the anterior chamber. This is shown experimentally by the injection of India ink into the anterior chamber, when particles of this substance are found very often in the tissue of the iris, especially about the veins, which had entered not only from the crypts, but also diffusely from the entire surface. I do not consider this sort of evidence convincing, and, even when we admit that secretion and resorption of the aqueous by the iris may be associated, this outlet for the aqueous through the vessels of the iris must be insignificant.

There are no other outlets from the anterior chamber; it is nowhere connected directly with lymph vessels, the aqueous escapes from the eye only by way of filtration into the blood vessels. *Leber* has shown that the absence of direct efferent lymph passages is of great importance to the preservation of the form and tension of the eye.

We do not know much with regard to the outlets of the vitreous. We do not know in what direction the fluid may move in the vitreous, or even whether a movement of fluid takes place. Experiments with injections of diffusible substances have as yet given us no information, except that it is known from the experiments of *Nuel* that a portion of the India ink injected into the vitreous, with care to avoid an increase of tension, flows out of the pupil. On the other hand, it has been demonstrated much more frequently, in such experiments, that the India ink extended backward into the sheaths of the central vessels and into the intervaginal space of the optic nerve. There are, therefore, two ways open for the flow of the vitreous fluid, backward through the optic nerve, and forward through the zonula.

Experiments to determine the quantity of fluid that escapes through the optic nerve have been made by *Priestley Smith, Leplat*, and *Niesnamoff*, in which fluid was injected into the vitreous under high pressure, while at the same time the outlet of the anterior segment of the eyeball was occluded by the introduction of vaseline into the anterior chamber, and have showed that not more than $\frac{1}{50}$ of the quantity of the fluid that escapes from the chamber leaves the eye in this way. The importance of this outlet is therefore very slight.

It has long been known that the vitreous fluid can filter easily through the zonule. I hold up by its optic nerve the eye of an ox which has had a little piece removed from the centre of its cornea. Drops of vitreous fluid continually come from the opening, and this fluid must have filtered through the zonule from the vitreous, as the aqueous has been removed. The same phenomenon takes place when the anterior chamber is reestablished in the cadaveric eye, according to *Deutschmann.* If fluorescin is injected into the vitreous and then the anterior chamber is punctured the coloring matter immediately exudes from the pupil in the living eye, hence it must have passed through the zonula.

It is unquestionable that this ability of the zonule in the normal eye to permit filtration is of great importance to the change of fluid, because no difference of tension exists between the anterior and posterior segments of the globe. If the tension in the vitreous is increased by the injection of fluid, that in the anterior chamber rises instantly. The iris and lens do not advance and the anterior chamber does not become shallow, as long as the eyeball is not opened. The equalization of tension between the anterior and posterior segments of the eye in this experiment must be due to the immediate passage of fluid through the zonule into the anterior chamber. Thus it is possible that the faculty of filtration possessed by the zonule is a sort of safety valve when pathological increases of tension take place in the vitreous.

The movement in the aqueous is very slow, but that of the fluid in the larger space of the vitreous is much slower. Indeed, it is doubtful if the aqueous secreted by the ciliary body enters the vitreous under pressure, for when we puncture the living eye, or irritate the ciliary body by subconjunctival injections of salt solution, we see substances that diffuse with difficulty, such as the albuminoids of the serum and certain antibodies, appear regularly in the anterior chamber, but neither *Wessely* nor I have found these substances in the vitreous. It is not at all certain that the renewal of the fluid of the vitreous does not take place through diffusion. The aqueous constantly secreted by the ciliary body mingles, in that case, with the fluid of the vitreous by diffusion through the zonule and the hyaloid membrane.

The perichorioidal space has obtained a certain importance through *Heine's* cyclodialysis for glaucoma. This is a capillary interspace between the chorioid and the sclera. Under normal conditions it forms at most an outlet for the tissue fluids of the chorioid, but not for the intraocular fluid, for the fluid would have to pass through the retina and chorioid in order to reach it, which is impossible. It is opened in cyclodialysis in the hope that a resorption of the intraocular fluid from the anterior chamber may be obtained. The outlet from this space is probably through the vessel sheaths of the venæ vorticosæ, either in *Tenon's* space, or, when these vessel sheaths end in cul de sacs within the sclera, as they are supposed to do, through filtration back of the fluid into the venæ vorticosæ themselves.

Whatever may be the details of the change of fluid in the eye a law of harmony must govern the process. It must be constituted so that secretion and excretion are evenly balanced to maintain intact the form, tension and function of the eye. We may epitomize this part of the theory of the change of fluid in the eye in the following manner: A gentle current of fluid passes through the eye so slowly that it cannot be seen, and regulated so wonderfully that secretion and excretion are exactly in equilibrium. How is this equilibrium disturbed in glaucoma?

125. The Nature of the Intraocular Circulation of Fluid

Before we can understand properly the pathological disturbances of this change of fluid, and how they are brought to light in glaucoma, we must discuss two important points: first the true nature of the intraocular circulation of fluid; second, the theory of the normal intraocular tension.

What are the powers that keep the intraocular change of fluid in motion? There is no doubt that the pressure in the blood vessels is the cause of the secretion and constant renewal of the aqueous and vitreous fluid. The best proof of this is furnished by the fact that after death, after the circulation has ceased, this secretion stops. A phenomenon, which, at first glance, seems to indicate that the secretion of the aqueous continues after death, is that if we puncture the anterior chamber of the eye of a cadaver it refills after a short time, but it has been proved that this fluid comes from the vitreous, filters through the zonule into the anterior chamber and is in no way a new secretion of aqueous. Evidently the difference of pressure between the anterior chamber and the vitreous, necessary for such a filtration in a punctured cadaveric eye, is produced by a slight compression of the vitreous by the elastic capsule of the eyeball, or by the retraction of the lens and zonule, which moved forward after the puncture.

Experimental stimulations of the sympathetic and trigeminus of dead animals by *Wessely* revealed nothing that indicates a new secretion of intraocular fluid after death. On the contrary the secretion is dependent on the existence of blood pressure.

From the remotest times the question has been discussed whether the process is one of secretion or of filtration, i.e., whether the living cells display an active secretory function, or the process is purely physical.

Query I.—Is the intraocular fluid produced by a process of secretion? Physiology teaches us to call a product a secretion only when it contains substances which are met with in the blood either in minute quantities or not at all, and when it is to a great degree independent of the blood pressure, as in the formation of the saliva, the gastric juice and the bile. Does the intraocular fluid correspond in its composition to such a definition of a secretion?

We have already studied the composition of the intraocular fluid, so I will simply state again that the aqueous and the vitreous contain only about 1% of solids, chief of which is sodium chloride, with small quantities of sodium carbonate, potassium chloride, potassium sulphate, calcium phosphate and magnesium phosphate, with traces of glucose and of paralactic acid, and a substance resembling mucin in the vitreous. The amount of albumin is very small, about $\frac{1}{40}$%, while the blood serum contains about 8%.

The most essential fact in this composition of the intraocular fluid is that it contains no material that is foreign to the blood serum. In addition to this the places where the intraocular fluid is formed, the ciliary processes and the iris, are not, correctly speaking, glands. Taking these things into consideration there can be no doubt that the intraocular fluid is not a secretion.

Query II.—Can the production of the intraocular fluid be explained by filtration? We speak of filtration when fluid passes, in consequence of a difference in pressure, through a porous membrane, by which the corpuscular elements suspended in the fluid may be detained, in accordance with the size of the pores.

We have two kinds of filtration, one through an artificial filter, the other through an animal membrane, which differ considerably from each other. For example, in the filtration of albuminous solutions through animal membranes the amount of albumin in the filtrate is considerably less than in the solution to be filtered, while this is not the case with an artificial filter. Filtration through animal membranes, i.e., through the membranes of the blood vessels, is the only kind to be taken into account in the formation of the intraocular fluid. It is also necessary to understand fully that we cannot apply the laws of simple, artificial filtration to the production of the intraocular fluid. The reasons we cannot do so are:

1. The filtration in the eye and elsewhere in the living body does not take place in the open air, as it does in our experiments, but between two hydrostatic pressures. The blood is forced into the capsule of the eyeball by the height of the arterial pressure, while the tension of the vessel wall and the intraocular tension press upon the blood column. The driving force for the filtration into the eye is the pressure difference between the height of the arterial tension on the one hand and the tension of the vessel wall, together with the intraocular tension, on the other.

2. Filtration never occurs alone in the eye, but is always accompanied by osmosis.

3. The living vessel walls, the layer of epithelium on the ciliary processes, and the endothelium of the iris, are not the same as simple, dry, dead, animal membranes.

Having made these reservations we can deal more accurately with the question: Can the production of the intraocular fluid be explained as a kind of filtration by means of the supposititious presence of very minute pores? To determine this we must answer two questions.

1. Can the composition of the intraocular fluid be explained by the theory of a process of filtration?

2. In how far can the quantitative relations of the change of fluid in the eye be explained by filtration?

As regards the first question, the salts of the intraocular fluid present no difficulty, especially as I believe that even their percentages scarcely differ from those in the blood serum, and it has been proved that the osmotic pressure of the intraocular fluid is not higher than that of the blood. All of these substances can certainly enter the eye by way of filtration, but how about the albumin? How does it happen that the aqueous and vitreous contain at least three hundred times less albumin than the blood serum?

Various causes have been adduced to explain the retention of the albumin. It has been believed that the intraocular pressure acted as a counterpressure to the filtration and made the passage of albumin difficult, but we see in intraocular inflammations, in which the tension remains the same, that it is unable to prevent the entrance of a considerable amount of albumin into the aqueous.

The epithelial covering of the ciliary processes and the endothelium of the iris have been thought to be the cause of the small amount of albumin that passes. I have already pointed out that the aqueous which reforms after puncture of the anterior chamber is very rich in albumin in comparison with the normal aqueous. *Greeff* found anatomically that after such punctures the epithelium of the ciliary processes becomes detached in the form of bullæ with degeneration of the cells, and supposed that this *denudation of the epithelium from the ciliary processes* was the cause of the greater passage of albumin, and also of the excretion of fibrin, into the anterior chamber. But this is not correct, for according to *Brauer's* researches, the escape of albumin when the tension is lowered suddenly is not associated with the formation of vesicles. Moreover, the formation of vesicles of the epithelium on the ciliary body is totally absent when the puncture is made very slowly and the tension is lowered gradually, and yet the amount of albumin in the aqueous is just as great as when the puncture is made rapidly. The epithelial blebs are likewise absent when we induce a great excretion of albumin into the aqueous through irritation of the eye, as by subconjunctival injections of salt.

The reduction of tension by the puncture cannot be the only cause of the increased escape of albumin. We would rather expect that with the sinking of the counterpressure the relative amount of albumin in the filtrate would diminish, in accordance with a law that has been discovered appertaining to the filtration of solutions of albumin. Instead of this we find just as much albumin in the aqueous in the course of inflammations and irritations of the eye, when the eyeball has not been opened, as after puncture.

If the cause of the retention of the albumin cannot be found in the counterpressure, nor in the layer of epithelium covering the vessels, there remains scarcely anything else, except to hold the vessels themselves responsible for the small amount of albumin in the aqueous. *In fact it must be laid down as an important law of the change of fluid in the eye that the escape of albumin into the aqueous is dependent primarily on the size of the secretory vessels.* We may convince ourselves of the extremely close connection between the condition of the vessels and the excretion of albumin into the aqueous by the following experiment.

We may brush the conjunctiva of a rabbit with a fine camel's hair pencil, or we may touch the eye with silver nitrate to obtain a greater degree of irritation, or we may make a subconjunctival injection of a 5% salt solution; in each case we will create a hyperæmia that extends to the secretory vessels. When the aqueous of animals that have been so treated is examined we find that the secretory organ of the eye has responded to the slightest irritation that produced hyperæmia with an increased excretion of albumin, which becomes enormous with the greater irritations. *Wessely* has shown that this is actually due to the abnormal size of the vessels.

When a powerful vasoconstrictor, like adrenalin, is used locally in the eye and then a puncture is made, the newly formed aqueous contains just as little albumin as the normal aqueous, because the vessels of the ciliary body are contracted.

Now we can return to our question: Can the slight amount of albumin in the normal aqueous be physically explained by the process of filtration? If so we must suppose that the pores between the capillary endothelia and the ciliary epithelia are so small that normally the large molecule of albumin cannot pass, but when these pores are stretched by hyperæmia the albumin passes through. Plausible as this theory seems, the fact cannot be concealed that the purely physical force of filtration does not suffice for the secretion of the aqueous, for we perceive signs in the eye which indicate that its secretory organ is able to keep back certain substances in the blood, in the same way as a gland.

When fluorescin is injected intravenously into a rabbit this easily diffusible substance is found in the normal aqueous, only in a much weaker concentration than in the blood. This is due, in the first place, to the fact that the organism begins the excretion of the coloring matter immediately after the intravenous injection, according to a pharmacodynamic law studied by *Loehlein.* Thus the prerequisite for a marked transition of such substances into the intraocular fluid is that they shall have been present in sufficient concentration in the blood for a long time, and this prerequisite is absent.

In spite of all these transmissions of drugs and coloring matters into the eye, vital cell functions play a part, and the simple law of filtration does not suffice to explain, for example, the observation of *Wessely* that after death the behavior of the intraocular process of secretion toward fluorescin is completely changed. Half an hour after death the fluorescin is no longer held back, in the manner above described, but passes freely into the anterior chamber, when the animal has been washed out with a salt solution containing the coloring matter.

Another evidence of the retention power of the secretory organ is that after the action of adrenalin only traces of fluorescin enter the anterior chamber even in case of puncture, as was found by *Wessely.*

The relations of the antibodies of the serum to the intraocular fluids furnish, in my opinion, still greater evidence that filtration is not the only factor in the secretion of the aqueous, but that vital elective powers of the cells take part. Under normal conditions these antibodies are held back in a peculiarly elective manner by the secretory organ. The albuminoids of the anterior chamber, small as is their quantity, do not differ, so far as we know, from those of the serum.

I have convinced myself by numerous experiments that the passage of the antibodies into the intraocular fluid, when the secretory organ is irritated, is by no means closely parallel to that of the albumin. As the albumin increases there is naturally an increase of the antibodies, but a percentage estimate shows considerable differences between them. When we puncture the anterior chamber of a rabbit that has been immunized against typhoid, whose serum contains a certain agglutination standard, for example, agglutinates typhoid bacilli in a dilution of 1 to 2000, and determine the agglutination power of the regenerated aqueous, we find that the aqueous with about 1% of albumin exhibits the same standard as the serum, although the latter has 8% of albumin. These things can be understood only when we recognize that the living cells of the secretory organ exercise vital functions in the production of the intraocular fluid.

We do not know precisely whether these cells are chiefly the ciliary epithelium, the endothelium of the iris, or the endothelium of the vessels. I advocate the view that capillary endothelia have to perform quite different functions in different parts of the body.

In how far can the quantitative relations of excretion and secretion of the intraocular fluid be explained by the theory of a process of filtration?

Many experiments on the dead eye with the filtration manometer have shown that the quantity of fluid flowing from the eye is proportional to the pressure of the injection at the particular time, i.e., that the outflow from the eye proves to be proportional to the passage of fluid through the animal membrane. The higher the pressure the more fluid flows from the eye; when it ceases the outflow stops.

The comparative experiments performed by *Niesnamoff* on the same eye while living and when dead furnish still better evidence. They demonstrate that such a proportion exists between pressure and quantity of outflow that we can picture to ourselves the evacuation of the intraocular fluid, its resorption, as in truth a process of filtration.

The anatomical construction of the eye is also such as to favor filtration. The sinus venosus *Schlemmii* is embedded in the firm tissue of the sclera, where it cannot be compressed by the intraocular pressure. It is connected, not directly, but laterally, with the efferent veins, so that the blood pressure in it is particularly low. The construction of the other outlets also favor resorption. The veins of the iris and ciliary body, which take part, are plainly held open by the turgor, or by the elastic tension of the tissue.

The only thing that remains to be noticed is that not only the outflow, but also the inflow of the intraocular fluid is in a way dependent on the height of the blood pressure, so that here also we may speak of a process of filtration. In this experiment the general blood pressure is either raised or lowered while the attempt is made to keep the intraocular tension the same, or the intraocular tension is changed without alteration of the blood pressure.

It has also been shown that the quantity of secretion is dependent on the intravascular and extravascular difference of pressure; for example, the intraocular secretion ceases when the secretory capillaries are subjected to an external pressure of 50 mm of mercury.

To recapitulate our views concerning the nature of the intraocular circulation of fluid we may say that the intraocular fluid is formed by a process of transudation, for its quantity depends on the blood pressure, and its quality on the chemical composition of the blood. The secretion of the aqueous differs from other processes of transudation, however, in the peculiar behavior of the passage of albumin, so the aqueous cannot be called intraocular lymph. In addition to this we must recognize that, extensive as is the physical dependence of the intraocular circulation of fluid upon the blood pressure, vital cell functions take part in the process of filtration. The eye is not a lifeless filter.

126. The Intraocular Tension

When one palpates his own eye he feels that the wall of the globe and its contents are subjected to a certain pressure, which we call the intraocular tension. We have learned from our cases of glaucoma that this can be increased, and from cases of phthisis bulbi, detachment of the retina, and perforating wounds, that it can be decreased. Now we need to learn how the intraocular tension behaves under normal conditions, and what we must know of it in order to understand glaucomatous diseases.

I. Elasticity of the Capsule of the Eyeball and Its Importance in the Preservation of the Form of the Eye

We may consider the eye hydrostatically as a nearly spherical, elastic capsule filled with fluid. The elasticity of the capsule is so great that the wall of the eye is stretched but very little when the intraocular tension is increased.

When fluid is forced under increasing pressure into a fresh eye the form of the ball changes very little. The increase of volume of the globe is found by experiment to be very slight. In *Koster's* experiment the pressure was increased from 19 mm to 70 mm, yet the increase of the cubic capacity of the eyeball was only 45 cm—only 7-1000 of the original capacity. The stretching of the capsule is not proportional to the pressure in such experiments, but always grows less as the pressure increases, because of the peculiar elasticity of the cornea and sclera, and particularly because the deviations of the cornea and sclera from the form of a sphere disappear more and more as the tension within the eye increases, so that the eyeball is forced to approximate the form of a sphere, and a sphere has the greatest possible cubic capacity with a given amount of surface. The shorter, sagittal diameter is somewhat lengthened as the intraocular tension increases, while the incurvation at the corneoscleral margin, due to the difference in the curvatures of the cornea and sclera, is lessened.

We can follow these changes on the cornea very well with the aid of ophthalmometric measurements. As the form of the eyeball approaches that of a sphere the radii of curvature of the cornea must increase. Under physiological conditions the vertical meridian of the cornea has usually the greatest curvature, but as the form changes under the increased tension it may happen that this physiological asymmetry disappears and the opposite meridian may become the most curved. Such an inversion of the physiological asymmetry of the corneal radii has been frequently found by many authors (*Pfalz*) in glaucoma, but on the whole the stretching and the change of form of the capsule of the eyeball when the tension is increased is very small, a phenomenon which is of interest in the pathogenesis of glaucoma.

The sclera alone supports the intraocular tension and preserves the form of the eyeball. The elasticity of the chorioid is much inferior, as is shown by the fact that if a little aperture is cut out of the sclera the chorioid will protrude at once through the opening, and it hardly needs to be said that the delicate retina cannot support the intraocular tension. *Nicolai* has found that the retina and chorioid are thicker when an enucleated eye is punctured previously to being placed in the fixing fluid. He believes that these membranes are kept stretched during life by the tension of the eye, and contract when that tension is removed. *Koster* thinks it more probable that a tissue tension exists in these membranes during life, which is equal to the intraocular tension and prevents their compression. When the tissue tension ceases with death the still existing, though lowered, tension of the eye may suffice to compress these membranes.

II. The Origin and the Height of the Intraocular Tension

The only source of the tension of the eye is the blood pressure.

If we divide the long ciliary arteries, which supply the ciliary processes—an experiment that has been performed by *Wagenmann*—the eye immediately becomes and remains soft. The blood is forced into the eye by the height of the arterial pressure against the resistance of the elastic wall of the eyeball, therefore the blood pressure must always be higher than the intraocular tension, for the blood column is subjected to both. The height of the arterial blood pressure must exceed the intraocular tension by the amount of the tension of the vessel wall.

The height of the arterial blood pressure in man is about 120 mm of mercury. The height of the pressure in the ophthalmic artery and its branches, which is of special interest to us, may be determined by pressing fluid into the eye and noticing the exact pressure at which the entrance of blood through the arteries ceases, or just begins again, and, according to the experiments of *Schulter*, the pressure in the ophthalmic artery in rabbits is almost as high as in the carotid, about 90 to 100 mm of mercury.

The pressure in the capillaries of the ciliary processes is, on the contrary, much lower. Its height is deduced from the secretion pressure of the aqueous, and is given by *Niesnamoff* as 50 mm of mercury, for with this height of pressure the secretion of the intraocular fluid ceases.

Height of the Intraocular Tension

We have at our disposal two means by which to determine the height of the intraocular tension, manometry and tonometry.

In manometry a canula is introduced into the eye, usually the anterior chamber, and connected with a suitable manometer. The experiment may be performed with *Leber's* filtration manometer, by means of which we may measure both the tension and the filtration.

Tonometry alone can be used for the examination of the human eye. An impression is made on the eye with a certain force by means of certain instruments, and the height of the tension is determined from the degree of force necessary to produce such an impression.

The intraocular tension of the human eye under normal conditions is about 25 mm of mercury, but this may vary within certain physiological limits, between 15 and 30 mm of mercury. We know that the eye is not simply a cavity filled with fluid, that it is divided by the iris and lens into an anterior and a posterior segment, that the vitreous space is filled with a gelatinous tissue, the anterior chamber with fluid. Experiments have proved that no differences of tension exist between these two cavities that can be measured with the manometer. If fluid is injected into the vitreous the tension in the anterior chamber rises at once to correspond. Likewise, no difference can be detected when one canula of the manometer is introduced into the vitreous and the other into the anterior chamber. Pulsatory and respiratory fluctuations of tension may be demonstrated in both spaces. Certainly, if a continuous flow of aqueous takes place from the posterior chamber into the anterior, as is assumed in the theory of the change of fluid in the eye, there must be a difference of tension between the anterior and the posterior chambers to overcome the resistance of the sphincter pupillæ, but we know nothing of, and cannot measure this difference of tension. At all events no great difference of tension exists between the anterior and the posterior segments of the eyeball.

III. Influences Exerted upon the Intraocular Tension

It has been known for a long time that certain fluctuations in the intraocular tension take place synchronously with the pulse and respiration, and recently they have been demonstrated with exactitude by *Wessely*. The pulsatory fluctuations are very slight, yet the changes in the size of the intraocular arteries suffice to excite them, although, under normal conditions, we can see no pulsation in the retinal arteries by means of the ophthalmoscope.

Influence of Drugs upon the Intraocular Tension

The question of the influence of drugs upon the intraocular tension is of special interest because we have to depend upon them in treatment, so a brief discussion of the pharmacology may be in order.

The four most important drugs that reduce the intraocular tension are adrenalin, cocain, eserin and pilocarpin.

Adrenalin, when used locally in the eye, whether by instillation, or by subconjunctival injection, reduces the intraocular tension because it contracts the blood vessels very markedly. This is the more interesting because the blood pressure is strongly increased by its intravenous injection. The enormous power of this drug to contract the blood vessels may be demonstrated by the instillation of a few drops of a 1 to 1,000 solution into the conjunctival sac of a patient.

The cocain that we constantly use likewise contracts the blood vessels and so reduces the intraocular tension slightly.

Eserin produces a similar but much greater effect. After a brief and slight increase the tension sinks very distinctly and for a considerable time. The effect is confined to the eye that is treated. *Groenholm* has shown by the filtration manometer that it decreases the rapidity of secretion, and also the plethora of the eye.

The effect produced by a 2% solution of pilocarpin is quite similar. At first there is a brief increase, followed by a decrease of the intraocular tension. Its effect on animals differs from that of eserin in that it is not confined to the one eye, but affects the flow of saliva and accelerates the pulse. Evidently it is absorbed more easily, for its effects are noticeable in the eye into which it was not instilled.

An important question in regard to the action of these two drugs is in how far their effect is due to the change they produce in the size of the pupil. We conceive the effect of meiotics in glaucoma to be that they facilitate filtration by making free the angle of the anterior chamber, but whether a dilatation or contraction of the pupil in a perfectly normal eye can influence filtration is doubtful. Probably the contraction of the blood vessels takes part.

Under certain circumstances atropin can increase the intraocular tension considerably. The filtration of the aqueous into the sinus venosus is rendered difficult, in glaucoma, by the dilatation of the pupil that follows its administration. The increase of tension seems to be preceded by a slight decrease.

Influence of the Nerves upon the Intraocular Tension

Only two nerves come under consideration: the sympathetic and the trigeminus.

Two phases follow each other in the intraocular tension when the sympathetic is stimulated: There is a brief increase, due in part to the contraction of the smooth muscles of the orbit, in part to a general increase of the blood pressure, or to the presence of vasodilator fibres in the nerve; then a more lasting decrease, due to the contraction of the intraocular vessels, a vasoconstrictor effect of the nerve.

When the sympathetic is divided the general blood pressure falls somewhat, and likewise the intraocular tension, but the effect on the latter is not great. It is remarkable that a reduction of the intraocular tension has been observed in man after extirpation of the superior cervical ganglion, a treatment that has been recommended in glaucoma.

Influence of the Trigeminus

Stimulation of the trigeminus causes immediately a very marked increase of the intraocular tension without any preliminary decrease, as has been found by *v. Hippel* and *Gruenhagen.* This increase of tension is due to an increase of the blood pressure, caused in part by the irritation of vasodilator fibres. *Adamueck* has proved that this increase of intraocular tension after irritation of the trigeminus is caused chiefly by the increase in volume of the blood vessels, but not by an increase of the intraocular fluid.

After section of the trigeminus the intraocular tension may sink, though this does not appear to be a regular symptom. Prolonged hypotony has often been observed in man after a paresis of the trigeminus, although keratitis neuroparalytica was absent.

The most important point to be learned from these results, so far as the theory of intraocular tension and change of fluid is concerned, is that these nerves have no direct secretory effect upon the eye, only an indirect effect by way of vasomotor influence. According to *Nicati,* after the trigeminus has been stimulated and the sympathetic divided, fluorescin that has been injected intravenously passes quickly and in large quantities into the anterior chamber after puncture, because the vessels are dilated. Inversely, the escape of the coloring matter is slight when the sympathetic is stimulated, because the vessels are strongly contracted.

The influence exerted by disturbances in the general circulation upon the intraocular tension is of interest because in glaucoma everything must turn on the question whether changes in the general circulation have brought about the disturbance in the change of fluid in the eye, or whether the latter is due to local conditions in the eye itself. The intraocular tension is directly dependent on the blood pressure, but this is no evidence that we can experimentally influence the former by decreasing or increasing the latter.

Compression or ligation of the carotid causes a reduction of the tension of the eye on the same side, but after a time the tension again becomes normal. The tension sinks when the vagus is stimulated, as well as after great loss of blood, and quickly falls at death.

If the spinal cord of an animal is divided between the occiput and atlas, so that the regulating action of the vasomotor centre ceases, the blood vessels of the abdominal viscera dilate extraordinarily, so as to contain a great part of the blood, and consequently both the blood pressure and the tension of the eye sink.

We can increase the tension of the eye by compression of the descending aorta, thus driving all the blood to the head. We can also increase it greatly by stimulation of the medulla oblongata, through stimulation of the vasomotor centre. The same effect is produced by carbonic acid poisoning, in which a stimulation of the vasomotor centre plays a part. We can also temporarily increase the intraocular tension by the intravenous injection of large quantities of physiological salt solution, but it lasts only a short time, in consequence of the rapid excretion of the fluid.

An obstruction to the venous outflow has almost no influence on the intraocular tension. If the obstruction is situated near the eye the blood evidently finds a sufficient way of escape through the dilatation of the vessels and through anastomosis. The only exception is that an increase of tension is produced by ligation of the venæ vorticosæ of the eye itself. Fluctuations in the intraocular tension may be produced by drugs that reduce or elevate the blood pressure. It has been observed to sink in the narcosis produced by chloral and to increase in that caused by ether. Scopolamin also increases the tension of the eye, probably through stimulation of the vasomotor centre and contraction of the external ocular muscles.

The tension is also considerably affected by changes in the atmospheric pressure.

IV. Regulation of the Intraocular Tension in the Normal Eye

It is important that we should know what guarantees the regulation of the intraocular tension in order that we may understand glaucoma. We may imagine that it is regulated in the following manner:

The intraocular tension is dependent on the blood pressure, yet it counteracts any sudden fluctuations in the blood contained in the intraocular vessels. If the blood pressure rises and causes an increased secretion of intraocular fluid, the intraocular tension rises and checks the increase of the secretion. If the blood pressure falls, so that the secretion of intraocular fluid, and with it the intraocular tension, is diminished, the outflow of fluid must be lessened as the result of the lowered tension. Therefore the intraocular tension is always striving to maintain a normal height. How far nervous influences take part in this regulation of the intraocular tension we do not yet know accurately. It is remarkable that the intraocular tension does not rise with the increase of aortic pressure that we meet with in chronic nephritis.

Doubtless the most important point in the pathogenesis of glaucoma is that we can produce in the eye a permanent increase of the intraocular tension only by the occlusion of the outlet of the intraocular fluid, as proved by *Leber* and *Bentzen.* We have therefore to take this obstruction of the outflow primarily into account in the explanation of the pathogenesis of glaucoma.

127. Speculations Concerning the Nature and Origin of Glaucoma

Thus far we have dealt chiefly with the clinical symptoms of primary glaucoma, but for scientifically speculative reasons we must inquire into the true nature of this circulatory disturbance of the eye. In spite of the enormous amount of industry and experiment, in spite of all the acumen employed, it must be admitted that the true nature of glaucoma is still unknown. I can give only a brief review of the most important theories that have been advanced concerning its fundamental causes. There is scarcely a tissue, scarcely a phenomenon in the eye that has not been held responsible for the origin of this disease. The following fundamental facts must be taken into account if we would become conversant with the theories of glaucoma.

The intraocular tension is etiologically dependent on the blood pressure and on the fullness of the vessels, and at the same time acts as a regulator of the intraocular plethora. If the blood pressure rises so as to increase the intraocular secretion of fluid, the increase of intraocular tension so produced counteracts the increase of secretion. Again, an increased secretion of fluid in a normal eye must result in an increased discharge, so as to restore the equilibrium between inflow and outflow. If the blood pressure falls and the diminution of the secretion causes a lowering of the intraocular tension, the discharge of fluid is lessened and the balance is restored. Another regulating factor, the importance of which was not formerly appreciated, is the elastic extensibility of the sclera. If an excess of intraocular fluid is actually present the healthy eye can accommodate itself to such a volumetric change of its contents by the stretching of the sclera.

The question now arises: By what can the intraocular tension be increased in pathological conditions, as we find it in glaucoma?

A. Retention Theories of the Pathogenesis of Glaucoma

We have to deal first with the fact that in animal experiments the intraocular tension can be permanently increased only by closure of the outlet of the intraocular fluid. These experiments have been performed in various ways. *A. Weber* injected olive oil into the anterior chamber; *Leber* produced an inflammatory agglutination of the sinus of the anterior chamber by the injection of dead cultures of staphylococci; others, like *Schoeler* and *Heisrath*, induced an increase of tension by cauterization of the limbus; *Bentzen* injected various substances, such as solutions of iodine and of ammonia, into the anterior chamber, cauterized the pigment edge of the cornea, and introduced threads into the anterior chamber, but succeeded in obtaining a prolonged increase of tension only by a severe injury of the sinus produced by scratching. A permanent increase of tension with increasing enlargement of the eyeball was produced in rabbits with greater precision by *Erdmann,* by the introduction of finely granular oxidation products of steel. This may be done by introducing a steel needle as a positive electrode into the anterior chamber, placing the negative electrode on the back of the animal, and passing through them an electric current of suitable strength. Such oxidation products may be produced by electrolysis in the aqueous which has first been evacuated from, and is then replaced in the anterior chamber of a rabbit. The finely granular particles of iron are deposited in *Fontana's* space, where they produce by cell proliferation a permanent occlusion of the sinus of the anterior chamber.

Animal experiments prove the importance of the occlusion of the sinus of the anterior chamber in the production of an increase of the intraocular tension, and occlusion of the outlet must be of great importance in human glaucoma, since it can be anatomically demonstrated in almost every case. This has been confirmed repeatedly since the researches of *Knies.*

In addition to this anatomical fact an experiment can be performed on the human eye. *Leber* and *Bentzen* investigated the filtration capacity of enucleated glaucomatous eyes and found that this capacity was greatly reduced, showing that the occlusion of *Fontana's* space may cause increase of tension. It can be understood from the consideration of such important facts in the pathology of the human eye how it is that many ophthalmologists have ascribed, and still ascribe, the origin of glaucoma to an occlusion of the outlet. They are the advocates of the retention theory. The question is only: What causes the occlusion of the sinus? A consideration of all the various theories that have been propounded in answer to this question would be going too far, so I will mention only the most essential.

Knies thought the cause to be a genuine inflammation in the vicinity of *Schlemm's* canal, and even now many, like *de Vries* and *Henderson,* suppose an engorgement to exist in the veins of the circulus venosus, a primary sclerosis of the fibrous elements of the ligamentum pectinatum. According to *Ad. Weber* a venous engorgement takes place in the ciliary processes in consequence of the reduction of blood pressure, which causes the ciliary processes to swell, and then the swollen ciliary processes force the periphery of the iris into the sinus of the anterior chamber. This is known as the *Weber-Knies* theory. According to *Ulrich,* glaucoma is due to a sclerosis of the connective tissue of the iris.

Others, like *Czermak* and *Birnbacher,* emphasize particularly the conditions found by them in the vortex veins as productive of the venous engorgement, while, according to *Priestley Smith,* a narrowing of the perilenticular space predisposes to the origin of glaucoma. It has already been mentioned that hypermetropic eyes are more prone to glaucoma than others. Hypermetropic eyes are smaller. We know that the lens increases in size throughout life, that it is a third larger at sixty five years of age than at twenty five, and that as it grows larger it approaches nearer, not only to the posterior surface of the cornea, but to the tops of the ciliary processes at the equator, thus narrowing the circumlental space, especially in eyes that are small, in hypermetropic eyes. In glaucoma the ciliary body is distended with blood, the ciliary processes are swollen and pushed forward. As long as the circumlental space is large enough these processes can expand into it, but if the space is small the ciliary processes are, according to *Priestley Smith,* driven forward against the periphery of the iris and occlude the sinus of the anterior chamber.

According to *Czermak,* the iris can easily cause an occlusion of the sinus of the anterior chamber, especially with its thicker portion, when the pupil is dilated. If the tension increases it compresses, at a certain height, the veins at the places where they enter the sclera and so produces engorgement and increase of transudation.

Still other ways of occluding the outlet have been thought of. Some ophthalmologists believe that obstruction to the outflow from the vitreous along the central vessels of the optic nerve may cause an engorgement of the intraocular fluid. *Laqueur* thinks that such an obstruction would be harmless as long as the anterior filtration space was open. Others have agreed with *Schwalbe* that the space between the chorioid and the sclera is a lymph sac, the distention of which may explain the symptoms of glaucoma.

Finally, the composition of the intraocular fluid itself has been held responsible for its retention. Some have believed that a chemical alteration causes an irritation of the endothelium lining the outlet passages, which proliferate and cause occlusion. On the other hand, many, like *Troncoso*, ascribe the difficulty of filtration to a large amount of albumin in the intraocular fluid, and the occlusion of *Fontana's* space to the excretion of albumin in the glaucomatous eye, but this is not correct, because no glaucoma need be produced by the albuminous aqueous in iritis. The anterior chamber may be entirely filled by a hypopyon without causing an increase of tension. The farther we get away from the occlusion of the sinus of the anterior chamber in this theory, and the more we bring in other factors to explain the retention, the more insecure becomes the ground beneath our feet. Although we recognize fully the importance of the occlusion of this outlet in the pathogenesis of glaucoma, yet, in spite of all efforts, the cause of this occlusion remains an enigma. No one can deny with certainty that the occlusion of the sinus of the anterior chamber is not a secondary result of some other process.

B. The Secretion Theory in the Pathogenesis of Glaucoma

Although in animal experiment we can produce a permanent rise of the intraocular tension only by occlusion of the sinus of the anterior chamber, yet it has long been known that there are other factors which are able to excite an increase of tension. It has been proved experimentally that such an increase may be excited by stimulation of the sympathetic and trigeminus. It is supposed that when these nerves are stimulated the blood pressure rises because of a contraction of the vessels and increases the secretion within the eye. Clinical experience also seems to indicate that the secretion may be increased by nerve irritation, as for example, when patients suffering from an obstinate trigeminal neuralgia are finally seized with glaucoma. An increase in the contents of the eyeball is also obtained when the venæ vorticosæ are ligated according to the procedures of *Schulten* and *Koster*. The rise of the intraocular tension in animals, associated with a change of the general blood pressure, has also been the subject of much study.

All investigators, who do not subscribe to the retention theory, continually insist that the occlusion of the outlet cannot be the sole cause of glaucoma, for occasionally we find the filtration space open when glaucomatous eyes are examined anatomically. Numerous clinical facts prove that partial occlusion of the sinus of the anterior chamber need not produce glaucoma. It may be occluded throughout a large extent by blood, hypopyon, and even marginal agglutinations of the iris to the cornea, and yet need not cause glaucoma for a very long time. Hence there are numerous ophthalmologists who consider occlusion of the outlet to be not the only cause of glaucoma, if the cause at all, but explain the rise of tension through an increase of the fluid contents of the eye. This brings us to the second group of theories: those of secretion.

Donders conceived glaucoma to be a hypersecretion caused by nervous influences, and the experiments of *Gruenhagen* and *v. Hippel* seem to show that irritation of the trigeminus takes part in its causation. The symptoms of acute glaucoma in particular, in which the tension suddenly rises, may be explained by an irritation of the trigeminus, according to *Gruenhagen* and *v. Hippel*, although disturbances in the circulation of the blood certainly cooperate. The irritation of the trigeminus is the more apt to result in a rise of the intraocular tension as the tonicity of the blood vessels governed by the sympathetic is the less intact.

According to *Schweigger* glaucoma is a neurosis of secretion which causes a volumetric increase of the vitreous. He calls particular attention to the fact that, according to the retention theory, we must observe, at least at the beginning of a glaucoma, a dilatation of the anterior chamber by the dammed back aqueous, but this is not the case.

Laqueur looked for the secretory nerves, the irritation of which would cause hypersecretion, not in the trigeminus, but in the sympathetic. *Adamueck* found that an increase of the intraocular tension could be produced when the sympathetic was stimulated, in consequence of the rise of the general blood pressure, and *Abadie* ascribed the origin of glaucoma to an excitation of the fibres of the sympathetic. A plexus of nerves in the middle of the iris should take a prominent part and from this *Abadie* explained the effect produced by iridectomy. First the trigeminus, then the sympathetic, is held responsible for the glaucoma, in the secretion theories, of which there are still other versions.

Some authors, instead of supposing that an active hypersecretion takes place under nervous influence, seek to ascribe the increased secretion of the intraocular fluid to inflammations and alterations in the vessels.

Inflammatory glaucoma was thought by *v. Graefe* to be a chorioiditis serosa. *Goldzieher* found an atrophy of the chorioid and of the choriocapillaris in glaucomatous eyes and conjectured that these lesions had caused a compensatory dilatation of the vessels of the ciliary body, and thus a hypersecretion. *Fuchs* supposes that in consequence of chronic inflammatory lesions in the chorioid the venous outflow from the vessels of the ciliary processes and of the iris is rendered difficult or prevented so that an engorgement with hypersecretion is produced.

Vascular changes not caused by inflammation have also been adduced as answerable for glaucoma. *Parent* and *Rochon-Duvigneud* lay principal stress on sclerotic changes in the retinal vessels, from which come a hypersecretion and an œdema of the vitreous. This œdema of the vitreous may be the primary cause of inflammatory glaucoma, and the occlusion of the filtration angle a secondary symptom. *Schnabel* likewise considers vascular changes primarily responsible for the origin of glaucoma. Spastic changes cause the hypersecretion. If, for example, the vessels of the iris contract they secondarily cause a distention of the vessels of the ciliary body and so a hypersecretion. If the vascular changes are in the posterior part of the eyeball, in the region of *Zinn's* vascular plexus, they cause at first a redness and opacity of the papilla, which later becomes excavated by the formation of cavities in the nerve tissue. Glaucoma simplex may be purely a disease of the optic nerve.

Not only vascular changes in the eye, but disturbances in the general circulation of the blood have been held primarily responsible for an increased secretion of fluid within the eye by *Sulzer*, *Terson*, and *Zimmermann*. General arteriosclerosis is the primary cause, according to *Bajardi*, *Frenkel*, and *Evers-*

busch. It may at least be seen from this brief review that the advocates of the secretion theories can bring forward a number of factors that deserve consideration in the study of the origin of glaucoma. The two theories have frequently been combined.

C. Loss of Elasticity of the Capsule of the Eyeball in the Pathogenesis of Glaucoma

We have still to do justice to a third group of theories, in which the condition of elasticity of the capsule is considered the primary cause of this disease. Now that we can prove, as has been done by *Leber* and *Pilzecker*, and *Uribe y Troncoso*, that the normal eye can accommodate itself to an increase in the volume of its contents, that the capsule of the eye stretches considerably without any increase of tension, the suggestion is obvious that the sclera has lost its elasticity in a senile eye that is attacked by glaucoma.

We see clinically in our operations that the influence of the sclera upon the condition of tension must be carefully considered. Sometimes we find that the iris is driven forward after the anterior chamber has been opened in a cataract extraction, sometimes we see the cornea sink back like a saucer on account of the loss of elasticity of the sclera. The loss of elasticity of the sclera was held responsible for the occurrence of glaucoma by *Cusco*, *Coccius*, and *Stellwag v. Carion.* A diminution of its diameter, a thickening, or a sort of fatty degeneration of the sclera has been found frequently in glaucomatous eyes. If the normal sclera exerts, under normal conditions, a regulative influence, in that it counteracts an increase of secretion when the arterial blood pressure is high, and at the same time accelerates the outflow, we may well imagine that when the sclera is sufficiently rigid the outflow of an engorging intraocular fluid may be prevented. Opinions differ again as to how the engorgement of blood occurs in individual cases.

Many investigators, like *Straub*, *Nicolai*, and *Vennemann*, believe that it is not the loss of elasticity of the sclera, but of the chorioid that is accountable for the rise of the intraocular tension. It is not the sclera, but the chorioid that has to bear the pressure of the vitreous and to disburden the venæ vorticosæ. If the chorioid loses its elasticity the pressure acts upon the veins and produces an engorgement in them.

When asked for my own view I must admit that I am convinced from clinical observation, as are most ophthalmologists, that a universal explanation of the varying picture of glaucoma is not possible. In general, I think that the change in the capsule of the globe, which has hitherto been held in much too slight estimation, is the primary cause and furnishes the predisposition to glaucoma. The change in the sclera has been so little appreciated only because it is so slightly prominent clinically and anatomically, yet changes, such as the loss of elasticity, have gradually taken place, in the course of life, which predispose to the clinically recognizable outbreak of glaucoma. When we see that disturbances in the regulation of the intraocular change of fluid are often present in eyes which have not this disease, even for long periods of time, without necessitating a glaucomatous disease of the optic nerve, it seems to me that the hypertony cannot be looked upon as, per se, glaucoma. Prolonged increased tension can cause mechanically excàvation and injury of the optic nerve. Therefore, without underestimating the occlusion of the sinus of the anterior chamber and the increase of the intraocular excretion of fluid, I believe that, in primary glaucoma, the increase of the intraocular fluid, the occlusion of the sinus of the anterior chamber, and the rise of the intraocular tension are secondary symptoms. The primary change in glaucoma and the predisposing condition for its clinical symptoms are to be sought, I think, in the capsule of the eyeball. If changes have taken place in the sclera near the sinus of the anterior chamber a condition is furnished which paves the way for an occlusion, in case of an increase of the intraocular fluid. If the change in the sclera extends to loss of elasticity of the lamina cribrosa it is evident that the disease of the optic nerve must be considered an integrant part of the disease glaucoma. If the loss of elasticity of the lamina cribrosa is particularly marked it can be understood how the excavation can be caused by a trifling increase of the intraocular tension. If a greatly increased secretion of intraocular fluid takes place from any cause whatever, the picture of acute inflammatory glaucoma will be produced, and it is a matter of importance in how far the sinus of the anterior chamber may be made patent, either spontaneously, by meiotics, or by operation. Only when we consider glaucoma to be a disease per se, which is perhaps caused by a degeneration of the capsule of the eyeball, and consider the intraocular tension, the increased secretion of fluid, and the occlusion of the sinus of the anterior chamber to be only secondary symptoms, can it be understood how we can influence somewhat the mechanical factors of the change of fluid by our therapeutic measures, but not be able to cure the disease as such very often

128. Hydrophthalmos Congenitus

The most striking lesion in this child is an enormous enlargement of all parts of the eyeball. The cornea appears to be not only abnormally curved, but also enlarged in all its diameters. One cornea is still transparent, but the other is diffusely opaque. The corneoscleral margin has become indistinct, and the entire sclera, so far as it can be seen, looks like thin, delicate porcelain, permitting the bluish appearing uvea to show through. The anterior chamber is extremely deep, the iris seems to be enlarged, looks dull and atrophic, and is tremulous, showing that the fastenings of the lens are loose, that the zonule is either stretched or defective. The intraocular tension is unquestionably increased. The nature of the disease is evident. We call it hydrophthalmos, but it is nothing else than a glaucoma, the glaucoma of the young eye. In true glaucoma, which is met with in elderly persons, the capsule of the eyeball is too rigid to yield to the intraocular pressure and to stretch, but when a permanent increase of tension occurs in the eye of a child the capsule of the eyeball may stretch considerably. This is the condition in hydrophthalmos. I will leave it undecided whether the capsule of the eyeball is perfectly healthy, or whether changes have taken place in the cornea and sclera, as seems probable to me, when it has been stretched so much, but the pressure which acts outwardly to distend the capsule, is also exerted inwardly upon the lens. This explains why the lens is the only organ in the hydrophthalmic eye that is not enlarged. It seems, on the contrary, to be much too small in comparison with the eyeball. The zonule may finally be torn by the stretching, and then we find in hydrophthalmos a luxation of the lens into the anterior chamber, or into the vitreous. In this child the lens is still in its normal place, it is still transparent, and additional evidence that the condition in the eye is that of glaucoma can be furnished by an examination with the ophthalmoscope, which reveals a very deep, glaucomatous excavation of the optic nerve. The peculiar opacity of the other cornea can also be recognized as glaucomatous. If the disease advances farther, or lasts for a long time, we meet with adhesions between the iris and the posterior surface of the cornea in the sinus of the anterior chamber.

The nature of this peculiar disease has not yet been sufficiently investigated, in spite of all the efforts that have been made. In the majority of cases there is a certain constitutional predisposition, for the enlargement of the eye may be distinctly marked at birth, or take place during the first six months of life. The lesion may be confined to one eye, but in over two thirds of the cases the disease is bilateral. We also find that several children in one family may be affected, and it cannot be accidental that hydrophthalmos is sometimes associated with a plexiform neuroma of the lids and orbit. Its occurrence is therefore connected with a degeneration of the ciliary nerves. In other respects the anatomical examinations that have been made of such eyes reveal chiefly lesions which have been caused by the glaucoma. *E. v. Hippel* found, in the examination of the hydrophthalmic eye of a child four weeks old, a large ulcer of the posterior surface of the cornea, which had caused the increased tension through cyclitis and lesions of the ligamentum pectinatum. I myself found in one case that *Schlemm's* canal had not been formed, and we can readily understand that glaucoma must ensue *when the most important outlet for the intraocular fluid is developed insufficiently, or not at all, in consequence of a congenital anomaly.*

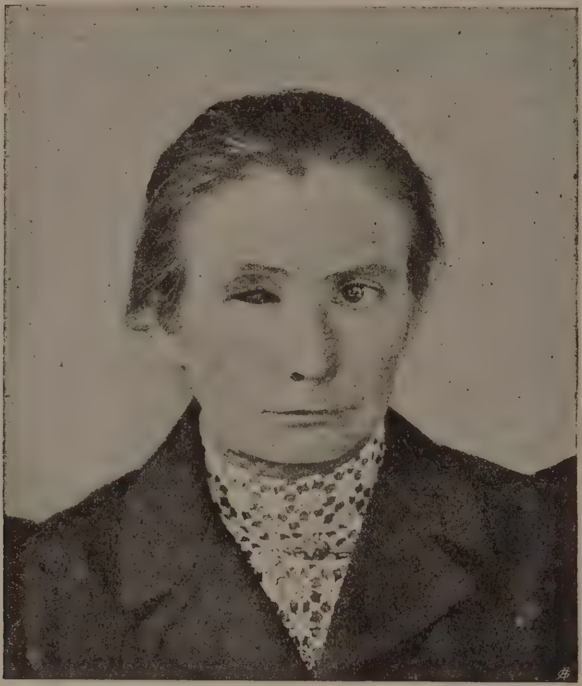

FIG. 103.—Left Eye: Hydrophthalmos. Right Eye: Anophthalmos.

The prognosis of this disease is bad. Such eyes may be ruptured easily by trifling injuries, and then they become phthisical. Even without such a traumatism the nature of the disease furnishes good reasons for the fear of a final amaurosis, though useful vision may be preserved for many years. The treatment depends on the individual case. I was able once to remove the lens, which had been luxated into the anterior chamber, and to save useful vision, but usually we try to secure this by conservative treatment, the use of eserin and pilocarpin, for experience has shown that the chances of the operative treatment of hydrophthalmos are not very good. When an iridectomy is performed the defective zonule may be easily ruptured by the escape of the great quantity of aqueous and the advance of the lens, so as to cause luxation of the latter, or a loss of vitreous. Sclerotomy has consequently been frequently recommended, but we must always consider it a stroke of good fortune if we are able to arrest the disease.

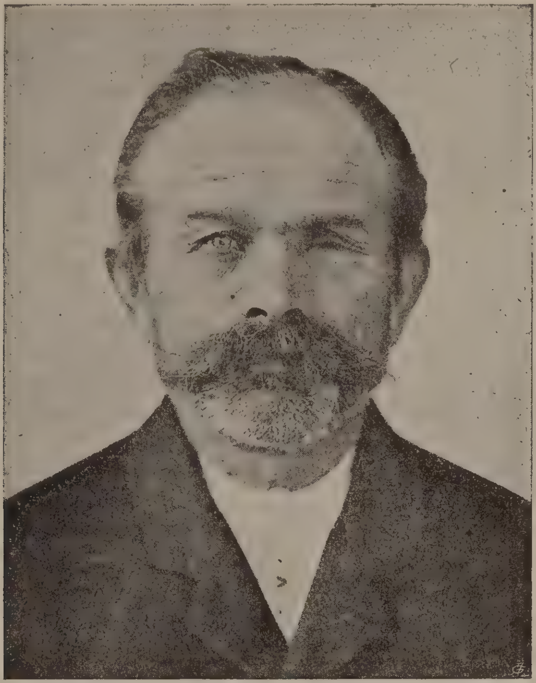

FIG. 104.—Right Eye: Normal. Left Eye; Anophthalmos.

129. The Commonest Forms of Secondary Glaucoma

It will be remembered that the glaucomatous conditions of the eye are divided into two large groups: primary and secondary glaucoma.

Primary glaucoma is a disease per se, which develops in the eye without the presence of any previous disease which might cause the increase of tension. The increase of tension in secondary glaucoma is the result of some other disease, and in the eye of this patient the increase of tension is plainly the consequence of a leucoma adhærens.

The glaucomatous excavation of the optic nerve can be seen with the ophthalmoscope in most cases of primary glaucoma, but in very many cases of secondary glaucoma conditions have been created by the previous diseases that preclude an accurate examination of the interior of the eye, so that we often have only three things on which we can depend for the diagnosis of secondary glaucoma: 1, an increase of the intraocular tension; 2, the presence of defects in the field of vision; 3, an impairment of vision which does not seem to be fully accounted for by the optic impediments. The test of the intraocular tension is the most important of these.

Another general characteristic of secondary glaucoma is that it remains confined to the one eye in which some other disease has caused the increase of tension, while primary glaucoma is accustomed to attack both eyes.

Increase of tension may be produced by, or occur in the course of, almost all serious diseases of the cornea, iris, lens, retina and chorioid. This is an important fact for both the general practitioner and the ophthalmologist. Over and over again I have tried to impress the fact that we must instinctively remember to test the intraocular tension in every serious disease of the eyeball. It is done in a moment and is worth the little trouble. It is our duty to obviate increase of tension whenever possible. If it is not done, or proves to be impossible, the secondary glaucoma goes on to blindness, the same as primary glaucoma, through excavation of the nerve and with the well known pain.

Although secondary glaucoma may follow almost all serious diseases of the eye, yet there are certain ones which exhibit an unmistakable tendency to this complication, while in others it is of rare occurrence.

Scars of the cornea that have become ectatic are to be taken primarily into account. Corneal cicatrices have scarcely any tendency to excite an increase of tension as long as they are flat, but as soon as one becomes ectatic, or is found to be so, we must apprehend secondary glaucoma. We meet with such ectatic cicatrices both with and without involvement of the iris. Even scars which become ectatic, when the cornea was not perforated and the iris is not involved, may excite increase of tension, but the latter is more frequently occasioned by corneal ectasiæ in which the iris takes part. Such is the case in this patient. The curvature of the cornea is irregular, because of an ectatic leucoma to which a portion of the iris is adherent. The anterior chamber in the vicinity of the adherent cicatrix is shallow, the iris is much distorted and atrophic from stretching, the tension is considerably plus.

The increase of tension in such a case can be understood. The conditions in the anterior chamber are no longer normal, the tension upon the iris extends to the ciliary body, the irritation of which leads to an increased secretion of the intraocular fluid, while the possibility of drainage is lessened. We perform iridectomy in leucoma adhærens with increased tension, and frequently we have to perform the operation in such a way that it will serve at the same time for optical purposes.

When other complications are present with a leucoma adhærens, such as a dislocated or swollen and wounded lens, the tension is more likely to become increased.

The harder and more unyielding the sclera, for example in old age, the less can the eye accommodate itself to the changed condition of tension and the sooner does the glaucoma appear, but a vicious circle is started by an ectasia of the cornea, even in young eyes. If the tension is increased as the result of occlusion of the sinus of the anterior chamber and irritation of the iris, the anterior segment of the globe yields more and more, the groove between the cornea and the sclera becomes obliterated, the eyeball acquires the form of an egg, the neighboring sclera appears bluish, and a sort of hydrophthalmos develops.

In cases in which the entire margin of the pupil becomes adherent to the cornea and a large staphyloma is developed after perforation of an ulcer, secondary increase of tension is produced directly.

Seclusio pupillæ is the first in rank among the diseases of the iris that lead to secondary glaucoma. In our next patient we find a circular agglutination of the margin of the pupil with the lenticular capsule, the result of several recurrences of plastic iritis. This is seclusio pupillæ. The middle portion of the iris is driven forward by the aqueous confined behind it, so as to form a crater pupil; the anterior chamber is shallow, the bulging iris almost touches the cornea; the increase of tension is distinct.

The origin of the secondary glaucoma in this disease is plain; the intraocular fluid, as it is secreted, can no longer pass through the pupil into the anterior chamber, but must accumulate behind the iris. This accumulation of fluid in turn causes further engorgement in the ciliary body and so an increased transudation into the vitreous. It may also be that the dragging on the nerves by the stretching of the iris contributes to maintain an increased secretion.

We may aid our patient by an immediate iridectomy, which is a most successful operation in chronic iritis and exclusion of the pupil. There is only one exception, which must not be forgotten. We occasionally meet with a stage of secondary glaucoma in sympathetic iridocyclitis when there is a total posterior synechia. We dare not perform an iridectomy in such a case, until the inflammatory symptoms have completely disappeared, for two reasons: first, the coloboma may be reclosed by a fresh exudate; second, a hypotony with subsequent detachment of the retina may easily be induced in such an eye.

Secondary increase of tension from isolated posterior synechiæ are much more rare, but such cases have been reported.

It frequently happens during the course of a serous iritis that the tension becomes elevated very much, as is exemplified by this patient, who is under treatment for that disease. Numerous deposits are to be seen on *Descemet's* membrane, the pupil is very widely dilated, there are no posterior synechiæ, the anterior chamber is rather deep, and the tension is distinctly increased. I prefer to perform a paracentesis of the anterior chamber in such cases, but it is also necessary to stop the atropin, which is ordinarily our main reliance in iritis, and to substitute eserin for a time. Cases have been reported in which grave secondary glaucoma followed a recurrence of a serous iritis. This disease has been named iritis glaucomatosa.

Certain diseases of the lens, particularly traumatic cataract and luxations, are apt to cause secondary glaucoma. Traumatic cataract has already been discussed, but I present this case because it exhibits distinctly the onset of secondary increase of tension. It is remarkable how apt the lens substance is to exert a bad influence on the tension of the eye by its swelling. The glaucomatous condition depends primarily on the rapidity of the swelling, which is not always proportionate to the size of the wound in the capsule. Every clinician knows that such lenticular substance is borne much better by one eye than by another, and that the rapidity with which it is resorbed varies greatly. It must also be taken into account in traumatic cataract whether the sinus of the anterior chamber is occluded or not, and whether the iris is pressed against the cornea. The iris itself reacts very differently in different eyes to the swollen lens substance; sometimes no synechiæ are formed; at other times agglutinations of the iris with the remains of the capsule and lens form very easily, even when the condition is perfectly aseptic.

An eye with traumatic cataract complicated by secondary glaucoma must be watched very carefully. If the tension rises, or the pain increases, we relieve the eye by evacuation of the swollen lenticular masses through an opening into the anterior chamber.

A case of subluxation of the lens presents an uneven depth of the anterior chamber, a tremulous condition of the iris, and visibility of the margin of the lens. Test of the tension reveals a distinct increase. Dislocations of the lens are often associated with increase of tension. We meet with this form of secondary glaucoma in both congenital and acquired dislocation, and we can say that a very large portion of the eyes that suffer from luxation of the lens finally undergo glaucomatous degeneration.

Numerous anatomical examinations have proved that sometimes the sinus of the anterior chamber is open in these cases, sometimes is occluded, and therefore we have to consider that both the occlusion of the outlet from the eye, and an increase of secretion caused by the irritation of the ciliary body can excite the increase of tension in secondary glaucoma from luxation of the lens.

The increases of tension occasionally met with after operations for cataract and secondary cataract belong among the secondary glaucomas produced by diseases of the lens. These we have already discussed.

The onset of secondary increase of tension is a symptom which occurs as a rule in the course of the development of intraocular tumors. I need only refer to those cases of detachment of the retina in which the diagnosis of a growing tumor was rendered possible by the increase of tension. All kinds of tumors may give rise to increased tension; sarcoma of the chorioid, tuberculosis, gumma, glioma, and the rarer tumors of the iris. The symptoms are those of glaucoma; engorgement of the episcleral veins, dilated pupil, cloudiness of the cornea, shallowness of the anterior chamber, and ciliary neurosis. It is important to know that secondary glaucoma may develop very gradually with failure of the vision, and also quite suddenly with the symptoms of acute glaucoma. A narrowing, or an obstruction of the filtration angle is found anatomically in most of these cases.

130. Rare Forms of Secondary Glaucoma

We distinguish for reasons of expediency between the common and the rare forms of secondary glaucoma. The physician has to deal with the common forms, as a rule, but for the sake of completeness I will utilize this patient as a text from which to speak of a few of the rarer ones.

A fairly large number of retinal hemorrhages can be seen in the fundus by means of the ophthalmoscope. They are situated for the most part about the posterior pole and, by preference, at the places where the vessels divide, are irregular in form, some are striated, and they vary a good deal in extent. The ophthalmoscopic picture was more distinct a few days ago, now the intraocular tension is increased. This is what we call glaucoma hæmorrhagicum, by which we understand a secondary glaucoma that developed sooner or later after the appearance of the retinal hemorrhages. Usually only one eye is affected, though cases have been reported in which the disease was present in both. Contraction of the visual field and excavation of the papilla do not appear until late in the course of the disease.

Our patient complains of moderate pain, but sometimes the symptoms are those of an acute attack; rarely the picture of glaucoma simplex prevails to the end. Most of the patients are elderly and show signs of an extensive arteriosclerosis. How the increased tension is brought about in these cases we do not know. We are confined therapeutically to the treatment of the arteriosclerosis and the instillation of meiotics. The hemorrhagic variety forms between 1 and 2% of all cases of glaucoma.

Another rare form of secondary glaucoma is met with occasionally in elderly myopes who have large posterior ectasiæ of the sclera associated with grave atrophic conditions of the chorioid. The excavation is remarkably less steep and less deep than is usual in other forms. The explanation given by *Graefe* for this is that the thinned sclera gives way before the intraocular pressure in high myopia, so that there is only a little difference in the level between the papilla and the surrounding sclera. Physiological excavations spread in such cases, and any cup that extends to the margin may be suspected of being caused by pressure. This form has been named by *Hirschberg* myopic glaucoma. The disease may run its course as glaucoma simplex, or as chronic inflammatory glaucoma with intermittent attacks. The treatment is as usual, meiotics and perhaps iridectomy.

Rarely glaucoma is met with in other diseases, in which we are, as a rule, unable to say whether there is an etiological connection between them, or whether the association is accidental. An attack of increased tension has been observed in several cases of retinitis pigmentosa. Although the intraocular tension usually falls after a detachment of the retina has lasted a long time, yet cases are met with in which severe glaucomatous symptoms develop after such a detachment. I am not speaking of the retinal detachments caused by tumors, which are very often associated with an increased tension as the tumor grows. Glaucoma has also been observed in atrophy of the optic nerve of cerebral origin. When in such a case a deep pressure excavation is already present the diagnosis can be made only by comparison with the other eye, in which the papilla may be atrophic without excavation. Increased tension has also been observed with choked disc and retinitis albuminurica.

Finally it may be mentioned that increased tension is sometimes met with in a number of congenital anomalies, such as coloboma of the iris, total irideremia, microphthalmos and coloboma of the optic nerve. Commencing opacities of the lens, enlargement of the ciliary processes, and rudimentary conditions of *Schlemm's* canal have led to the increase of tension in such cases.

131. Differential Diagnosis of Glaucomatous Excavation of the Optic Nerve

We have occasion to deal with three kinds of excavation of the optic nerve, which may be seen with the ophthalmoscope, the physiological, the atrophic, and the glaucomatous. The timely recognition of the last is the most important, for if a physician does not diagnose a genuine atrophy of the optic nerve as early as he should, the result is not so tragic therapeutically, because we are unable to arrest this degeneration, but, if we fail to recognize a glaucomatous excavation, or diagnose it too late, we have ourselves to reproach, because a therapeutic intervention was our acknowledged duty. It is well that we should consider its differential diagnosis, for sometimes it is not easy to recognize.

At first, when its development begins, there is no suspicious cupping of the head of the optic nerve. The papilla may still be of its normal color, or it may be somewhat redder than usual with a general indistinctness, and hemorrhages may be found upon it.

Congenital excavations are met with which are so deep and have such steep margins that they may be mistaken for glaucomatous excavations, but such cases are so rare as to be of interest only to the ophthalmologist. The differentiation is made by prolonged observation and by testing the vision. Exceptionally also we meet with a steep excavation in a genuine optic atrophy. The physician must bear in mind two different things in the differential diagnosis of the glaucomatous excavation, the fundamental differences between the three forms, and the way in which a glaucomatous excavation begins.

In our first patient, who has a physiological excavation, there is an unusually broad cup in the middle of the papilla, at the exit of the vessels, but it does not extend to the margin. This can be determined best from the behavior of the vessels. If these are traced backward, from the retina to the papilla, instead of from the papilla to the retina in the ordinary way, it will be perceived that they pass for a distance over the surface of the papilla before they bend down into the excavation. The consideration of the nasal side is of particular importance, where thick bundles of nerve fibres course over the margin. On the temporal side the cup extends nearly to the margin of the papilla, but if we observe closely the little vessels we will be unable to find a sharp bending of their courses, even if the physiological excavation reaches the margin at this point. The deepest part of a physiological excavation may appear to be pale, or even white, but the marginal portions retain their normal color.

If we now examine this patient, who has a glaucomatous excavation, we find that the cup extends to the margin of the papilla, that the tissue of the entire papilla is atrophic, and that the vessels bend sharply over the margin. The papilla is pale, of a greenish white, and nowhere presents a reddish color. The yellowish reddish halo glaucomatosus must not be mistaken for a part of the papilla, for if this mistake should be made the vessels would seem to run for a distance over the broadened tissue of the papilla before they bend sharply.

Difficulties multiply when the entire papilla is not excavated, as in this case, but a reddish crescent still exists on one side. Here we must observe closely whether a sharp bending of the vessels is present, we must test the tension and investigate the visual field. A physiological excavation also is usually bilateral.

Now let us examine this third case, one of genuine atrophy with an atrophic excavation, a marked condition of which is not as common as might be expected. In atrophy the nerve fibres disappear from the papilla while the lamina cribrosa retains its normal position, therefore the excavation is usually superficial and shallow, and the vessels slope gradually downward. It differs from the physiological excavation in that it extends to the margin of the nerve, like the glaucomatous cup, and in its loss of color. To differentiate glaucoma and atrophy ophthalmoscopically we look for the sharp bending of the vessels, and for the broad, yellowish halo glaucomatosus in glaucoma, while in atrophy the white scleral ring is rendered distinct. We also look for pulsation and the other signs of glaucoma.

The diagnosis of a commencing glaucomatous excavation is much more difficult than would appear from the study of these three cases. The excavation begins at the entrance of the vessels and in the temporal side of the papilla after the period in which the latter appears to be reddened and indistinct. The sharp bending of the vessels may appear first at the upper and lower margins of the papilla, but in the majority of cases the temporal margin is the first to be reached by the excavation.

132. Intermittent Ophthalmomalacia

This gentleman complains that, at irregular intervals, his eye becomes very sensitive to light and slightly painful. When he consulted me first the most marked symptom was a considerable softness of his left eye, which was so marked that the cornea appeared to be slightly wrinkled and the intraocular tension was estimated at — 3. Otherwise the eye was and is just as normal as the right. No signs are present of an intraocular inflammation. Now he is suffering from another attack; the softness of his left eye is not as extreme as it was on the former occasion, but it is very marked and easily to be felt. The condition is therefore one in which the intraocular tension of an otherwise perfectly healthy eye sinks periodically and remains subnormal for a considerable time. We designate this condition as intermittent ophthalmomalacia. It must be understood when such a diagnosis is made that all causes, that are demonstrable anatomically and clinically, which may produce reduction of the tension are absent. We meet in practice with such a reduction of the intraocular tension most commonly in phthisis bulbi, the outcome of serious injuries, or of intraocular inflammations, such as iridocyclitis and panophthalmitis. We also meet with a temporary hypotony in the course of various diseases of the eye, such as detachment of the retina and iritis, and frequently after a discission of a secondary cataract. None of these conditions are present in this case; the left eye otherwise is perfectly healthy, and yet we find in it changes which at least indicate the genesis of the reduction of tension. When the attack occurs the left eye presents a slight degree of blepharoptosis, a contraction of the pupil, and a slight degree of exophthalmos, symptoms that point to a paresis of the sympathetic nerve. The cause of the paresis of the sympathetic in this man is a swelling in the left infraclavicular region, known as a goitre, which evidently becomes swollen from time to time so as to press upon the sympathetic. When the swelling subsides the symptoms of this paresis disappear and the tension of the eye becomes normal once more. We have therefore to conclude that the sympathetic nerve plays an important part in the etiology of intermittent ophthalmomalacia. We learned, while speaking of glaucoma, that this nerve is of great importance to the intraocular tension, and yet there are cases of paresis of the sympathetic in which the softness of the eyeball is not perceptible. Therapeutically we may content ourselves with galvanization of the cervical sympathetic, if the goitre does not eventually render an operation necessary.

Aside from intermittent ophthalmomalacia there are cases that run a more chronic course, in which we frequently meet with other symptoms of an affection of the sympathetic; the eye may become considerably smaller and circulatory anomalies may appear in the affected side of the face. Finally, very rare cases of an apparently primary softening of the eyeball are met with, in which there are no demonstrable signs of trouble with this nerve. The pathogenesis of these cases has not yet been sufficiently investigated. I will take this opportunity to mention again that a high degree of hypotony may be observed in coma diabeticum.

XII

Muscular or Concomitant Strabismus

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133. Why Do Most Men Not Have Strabismus?

Binocular Single Vision and the Involuntary Effort of Fusion

A mother has brought her child to us because he is crosseyed; one eye turns inward. When I tell the child to look at this pencil it is quite plain that one eye looks at the object, while the visual line of the other is directed more toward the nose. The condition in which the visual lines of the two eyes do not intersect at the point of fixation is called strabismus. The mother naturally wishes to have this cosmetic disfigurement of her child's eye remedied. What should be done and advised in such a case?

In former times the treatment seemed to be fairly simple, to correct the position of the strabismic eye by operative measures, but, although the results were apparently satisfactory in many cases, yet the lame messenger followed after a while and we see now the less happy consequences of these hasty, and often improper, operations. At the present time an operation is the last resort, after every other possible method to improve the condition has failed, and our first study must be to determine the cause of the strabismus, in order to understand these other methods.

With the advance of our knowledge in regard to this subject it has been shown that scarcely one case of strabismus is like another, that the condition is not one simply of a faulty position of one eye, but that it is rather the external symptom of quite a number of various disturbances. A knowledge of the etiology determines what the proper treatment should be, but we shall have to go quite a way in order to answer the question, what has caused the strabismus in this child.

We must first learn why strabismus is not common to all men. The position of the two eyes is not ideal in man, but, on the contrary, adequate conditions are present to produce strabismus in everyone.

Try this experiment: Press a finger against the outer side of the eye; the entire visual field is immediately displaced and partial diplopia appears, because the equilibrium of the eyes has been disturbed. A slight mechanical displacement of one eye with reference to the position of the other suffices to interfere with the cooperation of the two and to cause strabismus. These are the conditions we shall deal with in spontaneous pareses of the ocular muscles. When there is a pathological lesion of the oculomotor apparatus strabismus must appear at the time the eye should be moved by the action of the paretic muscle. Certain irregularities also appear in almost all men, under normal physiological conditions, in the mechanical, anatomical forces that determine the position and movements of the eyes.

When we regard the features from the standpoint of the physician, certain asymmetries of the two sides of the human face can be detected, even in classic beauty. These asymmetries are met with particularly in the position of the individual eyes in the orbits, and the orbits differ in regard to their form, size and so called angle of opening. The eyes of individual men differ much in the form and size of the eyeball, as well as in its position in the orbit. This difference contributes greatly to the characteristics of the individual physiognomy.

Other important, though invisible, irregularities in the anatomical mechanical forces have to be taken into account. The cushion of fat differs in development, the muscles of the eyes differ in size, tendons and insertions, the fasciæ vary in development. In short, the position of rest of our eyes can scarcely be called ideal.

In the ideal position, which we term orthophoria, the visual lines of the two eyes are parallel, directed straight forward, and the vertical meridians are parallel. When we consider the origin of strabismus, our attention naturally turns first to this normal equilibrium of the eyes. If we wish to understand how and why the normal position of the eyes becomes disturbed, we must first learn the nature of the normal position. I wish to emphasize again that we can speak of orthophoria only when the eyes are directed straight forward by the mechanical, anatomical appliances in the orbit, and when the vertical meridian of the retina is upright.

Hanging on the wall is the so called tangent scale of *Maddox*, with a little electric light in its centre.

When we look at this light from a distance of several metres a retinal image is formed in each eye, but we see only one object with the two eyes, because the impressions transmitted from the two are blended in the brain so that we are conscious of only a single image. We can, however, easily change the form of the image in one eye so that it cannot be blended with the other, and thus produce double vision. We may do this by looking through a *Maddox* rod, which consists of a number of red glass rods placed close together in a metal frame. Each glass rod acts as a strong cylindric lens, which deflects the rays of light vertically to its axis, so that when we look through it the light seems to be drawn out into a long, narrow band. If we hold a *Maddox* rod horizontally before one eye and look at the light with both eyes, the light appears natural to the uncovered eye, while to the other it appears to be a long, red, vertical band. Thus we have obtained a double image, which we utilize in the test for orthophoria.

If the two eyes are in muscular equilibrium the red band passes exactly through the light seen by the other eye, but if there is a disturbance of the motor balance the band passes to the right or the left of the light. If we now place the *Maddox* rod vertically before the eye, the band will be horizontal and must pass through the centre of the light seen by the other eye if the equilibrium is normal. An imperfect motor balance found in this way is called heterophoria. The determination of the condition of equilibrium by the method of *Maddox* is based upon the suppression of the act of binocular single vision by an artificial alteration of the image seen by one eye. Various methods are employed to ascertain the condition of equilibrium of the eyes, but the tangent scale of *Maddox* is the easiest to use clinically.

We shall shortly come to the explanation why the tendency of the eyes to deviate from each other in their visual lines is first disclosed by this experiment, and why many people never become conscious of any disturbance in their muscle balance. Greater or less deviations from the ideal position of orthophoria are extremely common, they are present in most men. We must still take into account the fact that we have tested the muscle balance under only a single condition, therefore we can simply say that the two eyes are not situated ideally in their orbits, and that it would be expected from such a condition that most men would have more or less strabismus.

Why is it then that most men do not squint, in spite of these manifold differences in the mechanical factors that enter into the position of the eyes? The answer is: Because the position of our eyes with reference to each other is not determined wholly by the anatomical, mechanical factors that have been mentioned, but is regulated chiefly by the nervous influence exerted over the oculomotor apparatus, concerning which we have now to study.

The factors that influence the position of the eyes by way of the nerves are: 1, our will; 2, the tonicity of the ocular muscles; 3, the effort of fusion; 4, the association of accommodation and convergence. We do not need to analyze the first two factors, but the effort of fusion we must study more thoroughly.

Binocular Single Vision

I need not say that a man with two normal eyes sees surrounding objects singly, although both eyes receive the same impressions, but, self evident as this fact appears, the laws of binocular single vision are quite complicated. One of the many services rendered by the highly gifted *Ewald Hering* was the very exact investigation of these laws, from which we must content ourselves by selecting those that are of the most importance clinically. When we look into the distance with parallel visual lines all objects fixed upon at a distance of six metres or more are delineated on symmetrically situated places in the retinæ. The fixation point falls directly on the fovea of each eye and every other point falls on places in the retina that hold the same relative position to the centre. If we were to lay these retinæ one upon the other these places would cover each other, or coincide, so they are called identical points, or corresponding places.

The most important law of binocular vision tells us that the same kind of stimulation of both foveæ and of any other two corresponding places which occupy the same relative position to the centre of the retina, always results in a single sensation of sight. Objects which stimulate equally places in the retina that correspond, in this sense of the word, are seen singly by the two eyes. We call this phenomenon the sensory correspondence of the retinæ.

We will now note that under the influence of this sensory correspondence of the retinæ, when any object attracts our attention we direct our eyes toward it in such a way that it is delineated on our two foveæ, the places of sharpest vision. The movements are with each other when two corresponding places on the retinæ are stimulated and the visual lines simply have to change their direction, or against each other when the visual lines must be focussed for a greater or less distance in order to fix a disparately projected object. Another fact of importance is that the two eyes move in the corresponding manner even when one is excluded from the act of vision. If I fix upon this pencil and cover my right eye it remains directed toward the object, even though it is hidden.

The reason of this phenomenon is that the motor innervation of the two eyes is always uniform under normal conditions. According to *Hering* we may consider the two retinæ as parts of a centralized double organ, and certainly when two corresponding places on the retinæ are stimulated by one and the same object a single sensation is always produced. When two corresponding places are stimulated dissimilarly the different sensations appear in one and the same line of vision, and all objects that delineate themselves on corresponding places in the retina appear to be at the same distance as the fixation point.

On the other hand, stimulations of not corresponding, or disparate, places on the retinæ produce double images of the object in question. This is shown by an old experiment: If a pencil is held in the median line and the eyes are fixed upon it it is seen singly; interpose the forefinger of one hand in the meridian line between the eyes and the pencil, upon which the former continue to be fixed, and the finger will appear to be doubled; if the right eye is closed the left image of the finger will disappear, if the left eye is closed the right image will disappear, showing that the finger is seen in crossed diplopia. If we now fix upon the finger the pencil appears to be double, and when the right eye is closed the right image disappears, showing it to be seen in homonymous diplopia. We learn from this experiment that all points nearer the retina than the point of fixation create retinal images with crossed diplopia, and that the images of all that lie beyond that point give homonymous diplopia. This is because most objects are delineated on disparate places on the retina during vision with converging visual lines.

We differentiate between a horizontal and a vertical disparation of the retinal images; in the former the deviation from the position of correspondence is horizontal, in the latter it is vertical.

There are several causes why we do not notice these double images ordinarily when looking at near objects, under normal conditions. In the first place, the images of objects produced on the periphery of the retina are much more indistinct than those on the central portion, as may be demonstrated by looking straight ahead and moving a finger about in the periphery of the field. These images do not make as strong an impression on our consciousness as those on the fovea. We do not notice the things pictured on disparate places in the retinæ because our attention is directed wholly to the fixation point. In addition to this, even images which lie on corresponding places in the retinæ by no means always appear simultaneously in the proper line of direction. The images conflict with each other, so that we have what is known as the antagonism of the visual fields. One or the other image may appear alternately in the line of direction appertaining to the identical points. Finally, it has been proved that, within certain limits, objects may be seen with binocular single vision that lie on disparate places in the retinæ, but such objects appear to be nearer, or farther away than the point of fixation.

A physiological phenomenon, however, claims our chief interest at this time. We do not notice the double images because at the instant when our attention is directed to a disparately delineated object our eyes immediately and quite automatically execute such a movement as will bring the image upon the centre of the retina. To illustrate: If I hold a prism with its base out in front of my left eye and look at a light I see two lights for a moment, i.e., I have crossed diplopia, because the prism deviates the rays toward its base so that they fall no longer on the fovea, but on the retina at its temporal side. The diplopia lasts but a moment, because the left eye involuntarily turns inward, makes a movement of fusion, until the image falls once more on the fovea. The movements of the eyes that displace the retinal images from disparate to corresponding places are called fusion movements. These movements are in great part independent of our will and we call the power that produces them the involuntary effort of fusion.

The most important of these fusion movements are those by which the convergence is increased or diminished. Fusion movements can take place only when binocular vision is present; the involuntary effort of fusion acts only when there is not too great a difference in the perception of the images of an object received on two disparate places in the retinæ.

As soon as binocular vision ceases, or is impaired, as soon as the vision has sunk below a certain minimum, the involuntary effort of fusion is destroyed and, when a predisposition exists in consequence of anatomical incongruences, such an eye can assume the abnormal position of equilibrium given it by Nature, in other words, can take a position of strabismus. We very often see a strabismus develop in this way after the vision of one eye has been greatly impaired; it happens very easily because the involuntary effort of fusion is no longer in control, and very often because the mechanical, anatomical inequalities of balance are so common. When, however, binocular vision is intact and the power of fusion is present, a dominating force rules over the movements of the eyes, firmly overcomes the mechanical, anatomical anomalies of position, and so protects most men from strabismus.

Various methods are employed to test for binocular single vision, the importance of which will become evident. One of the most important and beautiful is *Hering's* test with falling bodies. Take a tube of black pasteboard with a cap at one end over which is stretched a fine thread having a small, black ball fastened in its middle; hold the tube before the eyes, look through it at the ball, and let little peas be dropped, some in front of, some behind the thread. A person who has two normal eyes can tell instantly on which side of the thread the peas fall, but if he closes one eye, so as to do away with binocular single vision, he will make many mistakes.

Another experiment is *Hering's* rod test. We take two little metal rods that differ in thickness; with both eyes open we can tell at once which is the nearer and which is the more distant, but when we use only one eye it is astonishing how greatly the perception of depth is impaired.

We have somewhat lost sight of our case of strabismus, and yet we have indirectly approached its cause, for we have seen that, of the four factors that influence the position of the eyes by way of the nerves, the binocular act of vision, the contact of the sensory with the motor apparatus, with the associated involuntary effort of fusion, is the most important. The question immediately arises: If this arrangement protects most men from strabismus, what is the condition in those who squint? Before we answer this question we must consider the second factor that enters into this problem, the connection between convergence and accommodation. The mastery of the experiments for testing the position of equilibrium, the faculty of binocular vision and that of the power of fusion forms the primary basis for the understanding of strabismus.

134. Why Most Men Do Not Have Strabismus

Connection between Accommodation and Convergence and the Relative Amplitude of Each

We have seen what an important factor binocular single vision is in the prevention of strabismus. We have seen that fusion movements constantly correct the anomalies in the position of the eyes caused by mechanical, anatomical factors and so protect most men, but we must study more closely the more important of these fusion movements, particularly those that take part in the development of strabismus. The increase and decrease of convergence and its association with accommodation form the second most important of the factors that regulate the position of the eyes by way of the nerves, and a knowledge of this factor is essential if we would understand why most men do not have strabismus.

When we speak of and study the movements of the eyes it must be understood that we are dealing with rotations about a point on the optic axis approximately 14 mm behind the surface of the cornea; the point of rotation therefore lies rather nearer the posterior than the anterior pole of the eye.

The primary position, from which the more important rotations of the eye take place, is that in which a point is fixed at infinite distance in a line of direction vertical to the frontal plane of the head. The visual lines normally are parallel in the primary position. Our eyes make both parallel, or associated, movements in the same direction, and symmetrical movements in opposite directions from this primary position. We look up, down, to the side, and in the oblique meridians. These are parallel or associated movements, but the movement that interests us in strabismus is the symmetrical movement of convergence. This we will now analyze, beginning with a pair of normal, emmetropic eyes.

We understand by the movement of convergence the power to direct both eyes at the same time toward one point. If we look first into the infinite distance, so that our visual lines are parallel, and then fix upon an object in the median line, both of our eyes make such a movement as will enable their visual lines to intersect at the point of fixation. We can perform this movement of convergence and binocular fixation only when we at the same time focus our eyes upon the point by means of a corresponding accommodation. There is, therefore, a definite connection between convergence and accommodation, a matter of very great importance in the etiology of strabismus, and we must try first to measure this connection.

The visual lines are parallel in their primary position, and when we move our eyes to fix on a point in the median line the visual lines form an angle with these parallels at the eye. This we call the angle of convergence. It furnishes us a measure for the movements of convergence performed by each eye. It is evident that the nearer the object in the median line is to the eyes the greater is the angle of convergence. The convergence in such a case is real or positive, and the angle of convergence is inversely proportional to the distance to the object. When the visual lines return from the object of fixation to a parallel position, i.e., when we look again into infinite space, the angle of convergence becomes zero.

Nagel suggested the conception of the metre angle as the unit of convergence. When we fix on a point in the median line one metre in front of the eyes our visual lines have a convergence of one metre angle. If we fix on a point one fourth of a metre distant we have a convergence of four metre angles. One advantage of this designation is that by it we are enabled to recognize still better the connection between convergence and accommodation that has been mentioned. An emmetropic eye, which is adapted to infinite distance when at rest, must accommodate 1 dioptre in order to fix on an object one metre distant, or 4 dioptres to fix on an object at twenty five centimetres; hence, in such a case, an accommodation of 4 dioptres is associated with a convergence of 4 metre angles. These 4 metre angles do not represent the totality of convergence, for most persons can fix with both eyes points that are much nearer. The totality of the convergence of a pair of eyes is called the amplitude of convergence. To measure this we must proceed in a way analogous to that in which we measure amplitude of the accommodation.

We have to know the far point and the near point of the eye in order to ascertain the amplitude of the accommodation. In the same way, in order to determine the amplitude of convergence, we must first find the point closest to the eye at which binocular single vision is possible, which gives us the maximum of convergence. Then we have to ascertain the most distant point at which binocular single vision can be maintained, which gives us the minimum of convergence. Just as the difference between the punctum remotum and the punctum proximum, expressed in dioptres, gives the amplitude of accommodation, so the difference between the maximum and minimum, expressed in angles of convergence, gives the amplitude of convergence of the two eyes. The formula for the determination of the amplitude of convergence is therefore similar to that used for the determination of the amplitude of the accommodation, $A=p-r$, except that we express the value of p and r in metre angles. The amplitude of convergence may vary greatly when the muscle balance is impaired, but we are now considering a normal pair of eyes. It has been proved that a normal pair has binocular single vision not only when looking at near objects, but also when looking into infinity, that is when the visual lines are either convergent or parallel. More or less divergence may also be associated with binocular fixation. The minimum of convergence is then negative, the far point is no longer in front of, but behind the eye, because the visual lines are divergent and intersect behind the eyes.

How can the amplitude of convergence be determined in practice? We find the maximum of convergence when we ascertain the nearest point at which the patient can fix with binocular single vision. We use for this purpose *Landolt's* ophthalmodynamometer, which consists of a simple tin tube, black outside, white inside, with a vertical, very narrow slit in its wall, which is placed over a burning light. The brilliant line of light stands out sharply from the dark background. A tape measure, divided into centimetres on one side, into metre angles on the other, is fastened to the bottom of the instrument. To determine the angle of convergence all that is necessary is to bring the instrument in the median line closer and closer to the eyes, which are fixed on the brilliant line of light, while the tape measure is held beside the temple of the patient. Finally a point is reached at which the line appears double in spite of all efforts to avoid it, and the images are crossed. The near point of the convergence, its maximum, is reached at this instant. We find the maximum of the positive convergence in this student to be 10 metre angles, i.e., he can fix with binocular single vision on an object until it is only 10 cm distant.

To determine the negative convergence, i.e., to ascertain how much the eyes can diverge without diplopia, we use an abducting prism. When a prism is held with its base inward before each eye the parallel rays of light are deflected toward their bases and strike the retinæ to the inner side of the maculæ, so in order to bring the rays upon the foveæ the eyes must correspondingly diverge. If the strength of these prisms is gradually increased we finally come to those that can no longer be overcome by the strongest effort. Binocular single vision is then abolished and diplopia is present. The strongest abducting prism that can be overcome in this manner indicates the maximum of the power of divergence of the eyes, the negative convergence. The point to which these strongest prisms deviate the rays is the negative far point of convergence; it lies, as I have said, behind the eyes. It would be very troublesome in practice to constantly change prisms of varying strength before both eyes, so we simply hold a prism in front of one eye. Both eyes must still diverge, because one eye cannot perform this movement alone, and we need only halve the angle of deviation of the prism in order to calculate the abduction for each eye.

All this can be obtained much more easily by the use of *Herschell's* double prism with *Landolt's* scale, which consists of two prisms of the same strength, that can be rotated in opposite directions with equal rapidity, so arranged that we can read on the frame the angle of deviation corresponding to each rotation of the prisms produced by a simple movement of the handle of the instrument, as well as the metre angle that corresponds to the angle of deviation. I hold *Herschell's* double prism before the right eye and begin to move it, while the eyes look at a light five metres away. At first a single light is seen, but soon

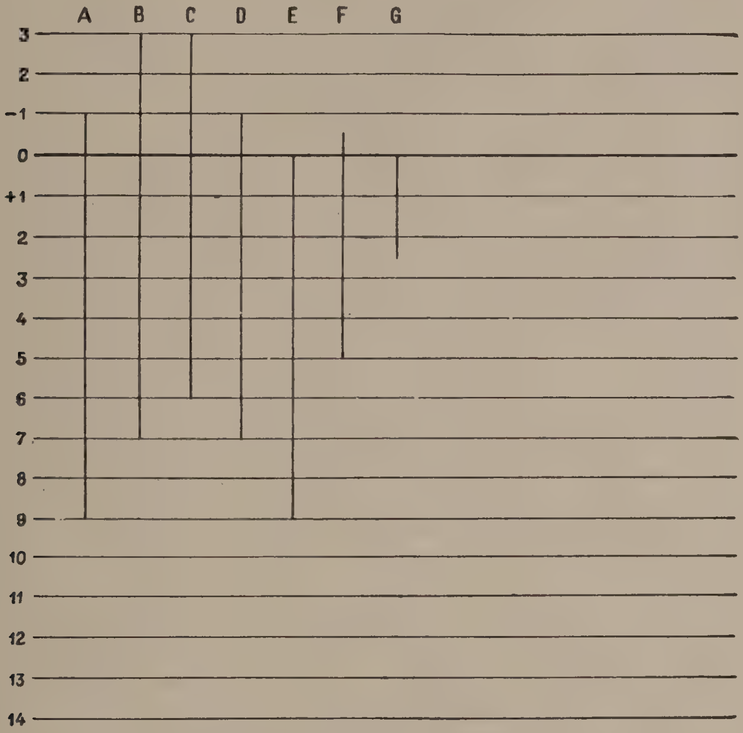

FIG. 105.—Amplitude of Convergence, after *Landolt*.

a position is reached which causes it to appear double. The eyes cannot diverge beyond this point and retain binocular single vision. We read the scale and find that the negative convergence amounts to 1 metre angle. The entire amplitude of convergence is therefore in this case, according to the formula, $a = p - r = 10 - (-1)$, 11 metre angles.

It is interesting to compare these symmetrical movements with the associated movements of the eyes. With a maximum convergence of 10 metre angles each eye is directed inward about 18°, while it can rotate to 30° toward the nose in the associated movement. With a minimum of convergence of 1 metre angle the eye is rotated out 1° 50′, but in the associated movement it can rotate outward 50°. Hence it follows that only a small part of the excursions that are made in the associated movements can be used in the symmetrical movements. According to *Landolt* we can graphically portray the amplitude of convergence in the following way: Let the line 0 represent the parallelism of the visual lines, the portion of the ordinates above the line the positive, that below, the negative part of the convergence. The numbers of the abscissæ give the metre angle. It is a good idea for students to determine the amplitude of convergence in each other's eyes, for in this way they gain a conception of its varying extent.

The relations between accommodation and convergence in hypermetropia are of the highest importance in the etiology of convergent strabismus. The majority of children whose eyes converge, are hypermetropic, and this leads up to another explanation of the fact that most men do not have strabismus.

We will suppose that we are dealing with a hypermetropia of 4 D. A person with that amount of hypermetropia must accommodate 4 dioptres in order to distinguish clearly an object in the distance. If the connection just described between accommodation and convergence were absolutely fixed his visual lines would have to converge 4 metre angles, would not be parallel for distant vision, but would converge and he would have diplopia. He would always find himself in the dilemma, either to see distinctly and double, or indistinctly and single.

Many emmetropes also, whose eyes have a rather divergent anatomical position, would fare badly if the connection between convergence and accommodation were absolutely fixed, for when such an emmetrope would render the axes of his eyes parallel by an impulse of convergence, he would be obliged to accommoderate at the same time and thus make his eyes nearsighted, so that he could not see well in the distance. The opposite condition prevails in myopia. The myope needs little or no accommodation for near work, and if he should not converge he would have diplopia and strabismus. As hypermetropia is the prevailing condition of refraction, many more men would have strabismus than actually do, if the binocular act of vision did not interpose in a special way as a regulator.

This regulative mechanism is represented by the relative amplitude of accommodation and convergence, by which we understand the power to increase

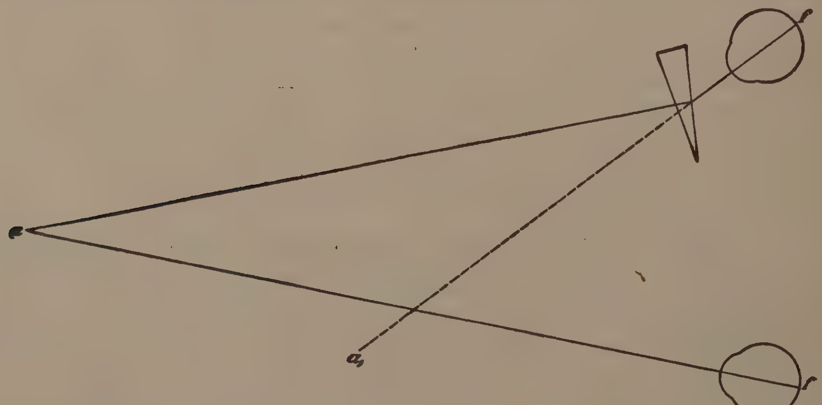

FIG. 106.—Experiment to Demonstrate the Relative Amplitude of Accommodation.

or decrease *separately* the innervation for convergence or accommodation, so far as may be necessary for the binocular act of vision. In other words: The connection between accommodation and convergence is not absolutely fixed, but is rather elastic. This relative convergence and accommodation may be shown by some simple experiments.

If a candle flame is looked at with binocular fixation and a prism with its base out is interposed before the left eye, the light is seen double for a moment, the images crossed, because the prism deviates the rays of light so that they fall on the retina of the left eye at the temporal side of the fovea, and the duplicate image appears somewhat to the right of the candle flame. This lasts only a moment, the light appears single again immediately, because the left eye, without our consciousness, rotates in sufficiently to allow the image of the light to fall once more on the fovea. If we hold the prism with the base in before the eye we have homonymous diplopia, because the image falls on the inner portion of the retina. We can fuse these double images also, up to a certain limit, because the eye makes a corresponding movement of abduction. This experiment and the accompanying drawing show that *the convergence of the visual axes may be diminished or increased while the accommodation remains unchanged.*

Another experiment is this: When concave glasses are placed before both eyes of an emmetrope and he fixes upon the candle flame, the glasses must be overcome by his accommodation. The connection between the latter and convergence would cause the visual lines to turn inward, so as to induce diplopia, but binocular single vision is able to partially sever the connection. *We can accommodate more or less with the convergence unchanged.* In this way it is possible to regulate in a suitable manner the association of accommodation and convergence. This is plainly an adaptation of the double eye to the needs and the difficulties of the act of vision created by its congenital or acquired anomalies. The principle of the adaptation is that the binocular act of vision makes these two innervations independent of each other. In hypermetropes, whose relative accommodation has to be excessive in order to enable them to see distant objects distinctly, the involuntary effort of fusion may prevent an overconvergence by an antagonistic innervation of divergence. In myopes whose accommodation is relatively too weak for the object of fixation, the involuntary effort of fusion may increase the innervation of convergence. Thus it is that although the combination of accommodation and convergence predisposes ametropes to strabismus, the tendency is overcome by the involuntary effort of fusion for a long time, sometimes permanently. The looseness of the connection between accommodation and convergence may be arrived at in another way. A hypermetropic child exerts the accommodation necessary for distant vision without exciting a simultaneous impulse to convergence of the eyes; it is only when he accommodates more strongly in order to look at near objects that the requisite innervation for convergence is added. Anyone who enjoys such studies can often find other signs which prove that the activity of these two innervations, that are apparently so closely connected, may be quite independent, but what has been said will suffice to elucidate the question why, in spite of many predisposing factors, most men do not have strabismus.

135. Heterophoria and Its Treatment

We have learned, in the preceding discussion, that imbalances of the eyes are demonstrable in many cases, under certain conditions, but that they are ordinarily obviated under the powerful influence of binocular vision through the involuntary effort of fusion. The tendency of the visual axes of such eyes to deviate from their normal positions does not become evident until the binocular act of vision has been rendered impossible. Such an imbalance, which is ordinarily concealed by the binocular act of vision, we call latent strabismus, or heterophoria. If students will investigate each other's eyes for its possible presence, they will be able to obtain an idea of what degree of imbalance can be borne without trouble, or may be designated as physiological.

People with hypermetropia complain of certain asthenopic troubles, but when a patient consults us on account of such an asthenopia we must not confine ourselves to the determination of the error of refraction, but always investigate the muscular balance of the eyes, for in many cases the symptoms depend on, or are maintained by, heterophoria. Look for heterophoria in all cases of asthenopia.

This patient complains that his eyes tire when used constantly for near work, that he has a feeling of pressure in his eyes, that the letters become indistinct, and that frequently objects look double. If, in spite of this, he keeps on working, he has headache and even nausea and dizziness. These are asthenopic symptoms, similar to those met with in hypermetropia, and this may be a case of accommodative asthenopia from hypermetropia, or it may be one of muscular asthenopia.

We may try to determine the cause in this case from the history. If the trouble is due to hypermetropia alone the symptoms do not disappear until the patient ceases from his near work, for if he closes one eye he must still continue to accommodate with the other, while, on the contrary, it is characteristic of a muscular asthenopia, due to a latent imbalance, that the symptoms disappear at once as soon as one eye is closed during near work. This is comprehensible, for in muscular asthenopia fusion compels the innervation to be increased sufficiently to conceal the latent imbalance of the muscles and the symptoms disappear when the patient closes one eye, because the involuntary effort of fusion is abolished and the strain of the excessive innervation no longer exists. We may therefore ask our patient whether he has ever tried to keep on working with only one eye, and, perhaps, let him make the experiment.

This, however, is not sufficient. The condition of the refraction must be investigated and the patient must wear the correcting glasses, for the relations between accommodation and convergence are disordered when errors of refraction are present, and the balance of the eyes may be influenced by them. A heterophoria that disappears after correction of the ametropia is called a pseudoheterophoria. If the asthenopia is not caused to disappear by the correction of the error of refraction we have to ascertain whether a latent imbalance of the muscles is present.

Two tests are useful for this purpose. One is to hold a pencil about 30 cm in front of the patient in the median line and have him fix his eyes upon it. If the muscle balance is perfect both eyes must remain properly directed toward the pencil, even when one is covered for a while, because in orthophoria the eye will remain in the position of fixation as the position of equilibrium is that which is produced by the correct innervation. How is it with our patient? When I remove the cover from before the eye we see it make a very distinct, rapid movement inward to the correct position of fixation. The eye makes what we call a *focussing movement* inward, hence it did not remain quiet when covered, but deviated outward. In other cases the focussing movement may be outward, upward, or downward. This test teaches us a characteristic symptom of latent strabismus; the eye makes a focussing movement the instant it is uncovered. In manifest strabismus, on the contrary, the strabismic eye makes a focussing movement, if it is still able to fix, as soon as the other eye is covered. We may conclude from this test that the eyes of this patient are not normally balanced during binocular fixation, but that they are compelled to maintain the position of fixation only by the binocular vision. It is only when we do away with binocular vision by covering one eye that the eyes show what is their real position of equilibrium, which in this case is a tendency to diverge. The eye deviates outward readily when covered because there is no longer any danger of diplopia when it is excluded from the act of vision. The eyes seek their natural balance because binocular vision does not compel them to overcome the imbalance. The visual lines no longer intersect at the point of fixation, but the conditions are right for crossed diplopia, and, in fact, the patient sees double at the instant the eye is uncovered, which suffices to excite fusion immediately.

We may imagine the entire trouble, in this case of latent divergent strabismus, to be that the internal recti are too weak in comparison with the external recti; the weak interni then receive a sufficient innervation during the instant of diplopia to counteract the tendency to divergence and to effect the necessary convergence. Latent imbalance is ordinarily not to be seen; it is only when one eye is covered, so as to abolish the impulse to fusion and render the excess of convergence useless, that the eye deviates outward to protect itself against weariness and to seek its true position of balance, which is not normal, but a certain degree of divergence. The assumption that one pair of muscles is weaker or stronger than its antagonists is, however, only a hypothesis; other mechanical factors may cause this motor disturbance; for instance, the insertions of the tendons may be abnormal. Purely nervous factors may also cause a group of muscles to be too strongly, or too weakly innervated. We are unable to assign a certain factor in the individual case, but for the sake of easy comprehension we will consider that in this patient the internal recti are too weak. Such weak interni could not receive continuously excessive innervation in order to maintain the necessary convergence for a long time without becoming tired, and then the trouble begins. The eyes are wearied by prolonged work, the letters begin to blur and frequently appear double; headache, and even nausea, follows. If we are to relieve these troubles we must make a second test. We must not be content with having found a latent imbalance, but must measure its degree accurately.

To do this we place the patient in front of a *Maddox* tangent scale, at a distance of 5 metres, and test with a *Maddox* rod. The test has been described already and it is only necessary to add that the large figures on the horizontal and vertical bars designate the degree of deviation at the distance of 5 metres. The figure through which the band of light passes gives us at once the degree of the latent deviation. We begin the examination by placing the rod horizontally before the right eye of the patient, so as to produce a vertical band of light and to determine first whether there is any latent horizontal deviation of the eyes.

It is desirable now to take note of a law we encounter in dealing with the muscles of the eyes. *Pathological positions of convergence result in homonymous, pathological positions of divergence in crossed double images.* We meet with this law in the test for latent imbalance.

If the *Maddox* rod is placed before the right eye and the red band appears to the right of the light, there is homonymous diplopia, which proves that the eyes have a latent tendency to convergence. This is known as *esophoria.* If the red band is to the left of the light the diplopia is crossed, proving a latent tendency to divergence, which we call *exophoria.* Our patient has an exophoria of between 7° and 8°, as is shown by his statements. If the rod is now placed before his left eye the condition will be reversed, but the same kind of diplopia, and the same degree of heterophoria must appear.

After the horizontal balance of the eyes has been tested we investigate their vertical balance. We place the rod vertically before the right eye so that the band will now be horizontal. If the band is below the light it shows that the right eye has a tendency to rotate upward, in comparison with the other. If we then place the rod before the left eye the band appears the same number of degrees above the light. Such a condition, in which the eyes have a tendency to assume opposite directions vertically, we call *hyperphoria*. We call the eye that deviates upward the hyperphoric eye, and in this case we say that the patient has right hyperphoria. The latent inclination of the visual axis below the horizontal line we call *cataphoria*. Finally we have a *cyclophoria*,[1] in which one or both eyes have a tendency to rotate about their sagittal axes.

To complete the test we investigate the heterophoria for near, using *Maddox*' small scale, in the centre of which is a little vertical rod. We hold the scale at a distance of 25 cm in front of the patient, place before his right eye a prism having an angle of deviation of 6° with its apex upward. He now sees the scale doubled; with his left eye he sees one rod below, while the one seen through the prism is displaced upward and we need only to note what number is indicated by it. If the lower rod moves to the left the diplopia is homonymous, if to the right it is crossed. We thus learn at once the nature and the degree of the latent deviation for near.

Treatment of Heterophoria

What shall we do in this condition? Too much is often done, frequently too little, so it may be well for me to give some therapeutic suggestions. This is work for the specialist alone.

Hyperphoria is the form of latent imbalance which is most productive of trouble. Patients with hyperphoria frequently complain of frontal pain and of a feeling of giddiness, and the physician should at least think of this possibility when such complaints are made, particularly of frontal pain. To correct this imbalance we order a prismatic glass of the strength found on the scale, with its base down in front of the eye that deviates upward. If the deviation is more than one or two degrees, so that the prism is heavy, the amount may be divided between the two eyes, the prism over the other eye having its base up, but if the hyperphoria is 3° or more we have to consider whether a tenotomy of the superior rectus is not indicated.

If we find a latent tendency to convergence we investigate first for hypermetropia, for, as every hypermetrope must accommodate strongly when looking at a distance, he must excite an innervation of convergence which is not opposed by the involuntary effort of fusion. If this involuntary effort of fusion has been excluded by the test with a *Maddox* scale, and a tendency to convergence has been brought to light, the first thing we have to decide is whether this tendency is not due wholly to the strong accommodation. Therefore we place the correcting lenses before the eyes of a hypermetrope and then test again. If the esophoria persists in spite of the correction of the hypermetropia, or if the patient is not hypermetropic, we may generally assume that the cause of the latent imbalance is anatomical and not situated in the nervous mechanism. Then we test the abduction of the eye and determine its amplitude of convergence. The negative convergence, the ability of the eyes to diverge, is of special interest in such a case, for when the ability of the eyes to diverge and the abduction are good, esophoria causes little or no trouble. If the ability to diverge is imperfect we prescribe prisms with bases out over both eyes to counterbalance the defect. I have sketched here the action of prisms in esophoria, showing how they permit the binocular fixation of an object in spite of the relatively strong convergence of the visual lines. We do not prescribe prisms stronger than 3°, because the glasses are too heavy, and because the stronger prisms produce other effects that are annoying. This limit serves for all forms of heterophoria. If the esophoria is over 8° for distance, operative intervention must be considered, tenotomy of the internus when the abduction and ability to diverge is normal, advancement of the externus when these are subnormal.

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[1] The description given by *Savage* of the innervation brain centres that maintain the upright position of the vertical axes of the eyes, together with an account of the way in which cyclophoria can be detected, measured and treated, will be found in the Appendix.—*F.*

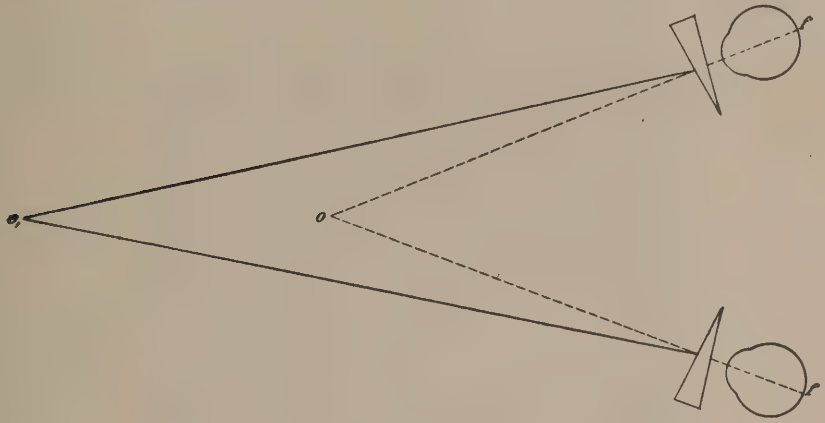

FIG. 107.—Treatment of Latent Convergence.

The form of heterophoria exhibited by this patient is the most common, latent divergence. We have to note the following points in regard to this condition.

Exophoria may exist alone, not associated with any other faults of the muscle balance, in which case it is slight and causes no trouble, or it may be combined with an insufficiency of convergence, when asthenopia is usually caused. We also meet with an insufficiency of convergence associated with a very slight exophoria, or none at all; in such persons we may find no heterophoria for distance, but as soon as we try the test first described, to cover an eye during fixation of an object, the other eye deviates outward.

Another test is to have the patient look at the point of a pencil held in the median line, which is gradually brought closer and closer to the eyes, until one eye is seen to deviate outward. The convergence is insufficient in such cases, not because of a weakness of the internal recti, for they do good service in adduction, but because of a weakness of the innervation for convergence. We often meet with such an insufficiency of convergence in anæmic persons. Improvement is frequently obtained from invigoration of the general condition, but it is advisable that systematic exercises be instituted in all cases of pure insufficiency of convergence. Errors of refraction are to be corrected. The patient reads at first holding his book at a considerable distance, draws it closer until the letters begin to blur, and then pushes it away again. We use prisms, with their bases in, occasionally in exophoria and insufficiency of convergence. As may be seen from this drawing, the rays of light coming from the point of fixation are deviated toward the base of the prism, so that the eyes need to converge enough to fix upon an object at a greater distance. Theoretically, therefore, these prisms can aid the convergence in latent divergence, or insufficiency of the interni, but they are not very useful therapeutically in practice. *Landolt* and *Monoyer* have proved by the determination of the amplitude of accommodation that an average of at least one third of the entire amplitude must be held in reserve, and only two thirds can be used when near work is to be done without trouble. In an analogous manner *Landolt* maintains that the 9 metre angles of positive convergence at the disposal of the normal man are in no way superfluous. Two thirds of the positive convergence must also remain in reserve, on the average, if asthenopic trouble is to be avoided during constant near work. From the calculation of the absolute degree of the metre angle, which must vary with the distance between the points of rotation of the eyes, it appears that an insufficiency of 2 metre angles requires a prism of 3°, but such a prism is too thick to be worn; the strongest we prescribe is one of 2°.

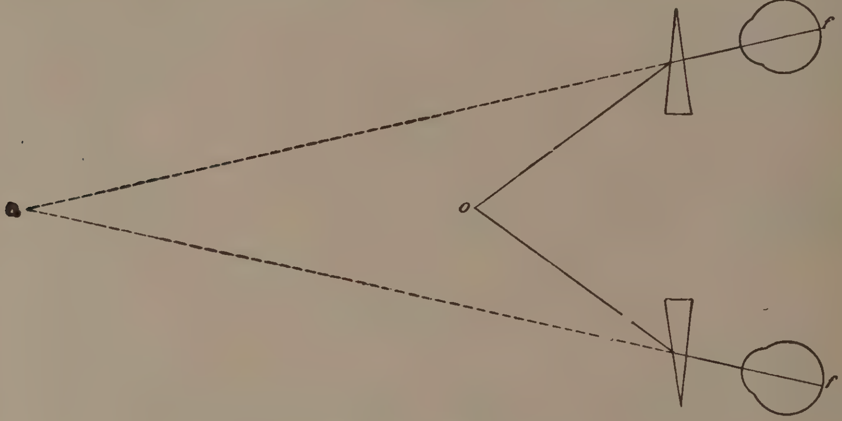

FIG. 108.—Treatment of Latent Divergence by Prisms.

I will repeat the steps to be taken in the examination of every case of heterophoria. I. Determination of the refraction and correction with glasses. 2. Determination of the nature and degree of the deviation on the tangent scale. 3. Determination of the amplitude of convergence; the maximum convergence by the aid of *Landolt's* ophthalmodynamometer, the minimum with *Herschell's* double prism, the record of the amplitude to be made on *Landolt's* blank. 4. Test of the power of adduction and of abduction. Investigation will convince anyone that many people overcome physiological heterophorias in the interest of binocular vision without trouble, or strain, and that in others they give rise to serious asthenopic troubles.[1]

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[1] [Credit should be given *Stevens* of New York, a pioneer in the study of the ocular muscles, not only for the introduction into literature of the descriptive terms heterophoria, exophoria, hyperphoria, etc., which have been adopted throughout the world, but also for the invaluable service he has rendered in the investigation of these conditions, and for the delicate instruments he devised, such as the phorometer, tropometer, and clinoscope, which were the first to render accurate detection and measurement of the various forms of imbalance of the ocular muscles possible. A description of the instruments mentioned and of the ways in which they are used will be found in the Appendix written by the translator.—*F.*]

136. Clinical Examination of Strabismus Convergens

The history of this case is quite characteristic. The parents noticed no deviation of the eyes during the first two years of the child's life, but they observed that one eye began to turn in toward the end of his third year. At first he was crosseyed only at certain times, at other times his eyes were straight.

During the stage of development of a strabismus when the deflection of the eye is not constant we call the condition *periodic convergent strabismus.* This condition may be the forerunner of a constant convergence of one eye. Strabismus is monocular from the first in about 80% of the cases, while in about 15% sometimes one and sometimes the other eye turns in and we have an *alternating strabismus.* The parents have frequently made good observations in this form of crosseye before they bring the child to the physician.

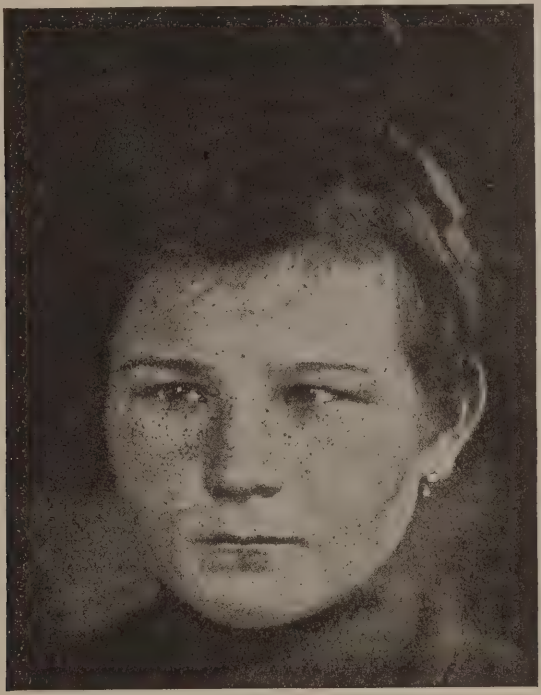


FIG. 109.—Left Eye: Strabismus Convergens.

Strabismus convergens usually develops in the early years of life. Statistics show that the monocular variety begins in about 75% of the cases toward the end of the fourth year, and that the alternating variety appears rather earlier in 30% of the cases, sometimes as early as the second year.

The objective examination commences with the determination of the vision, of the refraction with the skiascope, and of the corneal astigmatism by means of *Javal's* ophthalmometer. We often have to deal with very little children, and then we have considerable difficulty in ascertaining the vision. We take such children into the clinic and study them. We can tell from the way in which they run after *Worth's* ivory balls whether they see them, or are searching for them. Picture test types are also useful in the test of vision of little children.

One characteristic of the squinting eye in a monocular strabismus that has lasted a considerable time is the fact that its vision is poor. This fact is of importance for two reasons, one of which is therapeutic. If we wish to treat such a case of strabismus properly we have to try to improve and to cure this amblyopia. The other reason is that the amblyopia aids us in the understanding of the etiology and development of convergent strabismus. We shall consider this point later.

The determination of the refraction accompanies that of the vision in every examination. We ascertain it in the usual way, by means of skiascopy, and then instill atropin, in order to exclude the influence of the accommodation for a subsequent, more accurate, examination. Another fact of importance to the conception of the nature of strabismus convergens is that *the great majority of persons who have convergent strabismus are hypermetropic* and frequently have hypermetropic astigmatism.

The third question we have to answer in early cases of this nature is whether a strabismus really exists. There can be no doubt in such a case as this, but at first the convergence appeared only periodically, as has been stated, and in some cases a strabismus may not be real, but only apparent. I have here the photograph of a man. Everyone would say that he must have strabismus, yet he has not. The visual line from the fovea to the object of fixation does not pass through the centre of the pupil and of the cornea, but rather to its median side, and forms an angle, the angle γ, with the geometric axis of the eye, drawn from the posterior pole to the centre of the cornea. Hence we do not look directly forward, but obliquely, with our eyes. The size of this angle differs in different individuals. It is larger in hypermetropia, smaller in myopia. When it is very large a divergent strabismus may be simulated when the visual axes are actually parallel; in fact it may conceal a slight, real convergent strabismus. The visual line passes to the outer side of the apex of the cornea in rare cases, and then it is possible for a convergent strabismus to be simulated, or a divergent strabismus to be overlooked.

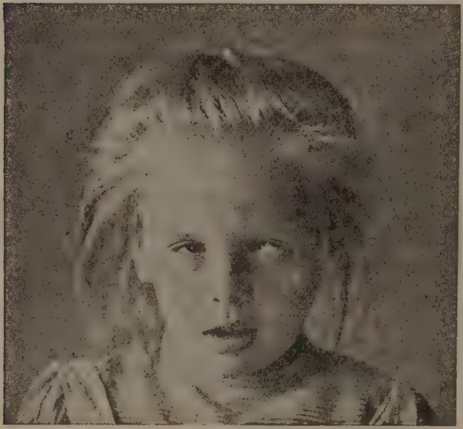


FIG. 110.—Strabismus Convergens: High Degree of Amblyopia.

We have recourse to a simple experiment to determine whether manifest strabismus is really present in doubtful cases, and when the degree is slight, which I may try on this patient, whose left eye turns in. I direct him to look at the crossbars of the window, which he does evidently with his right eye; I then cover his right eye and the other one immediately moves outward and assumes the fixation, but his right eye has turned in. This child fixes sometimes with one eye, sometimes with the other, and has what we call strabismus convergens alternans.

The most important point elicited for us by this cover test is that a manifest strabismus becomes visible the moment one eye is covered. The deviating eye takes command at this instant, by what is readily seen to be a focussing movement, provided it is able to fix. A latent strabismus, on the contrary, which is ordinarily obviated by the binocular act of vision, is revealed by the fact that the focussing movement of the covered eye takes place the moment the cover is withdrawn.

Another method of determining whether manifest strabismus is present is to throw light into the eyes by means of the mirror of the ophthalmoscope. Every child will look instantly into the mirror, older patients have to be directed to look at the aperture, and then we see a small reflection of the mirror on the cornea of one eye.

As the visual line does not pass through the centre of the pupil the image of the mirror is somewhat to the inner side of the centre. Now we pass the light quickly to the other eye. In binocular fixation the two images must be situated symmetrically, but if the visual line of one eye deviates from its proper course, if the eye is strabismic, the unsymmetrical position of the images will be perceived at once.

The focussing movements of the eyes are perceptible also through a modification of the mirror test, in which one eye and then the other is covered alternately during the experiment.

We may even go a step farther and determine approximately the degree of deviation of the strabismic eye from the position of the reflex images. It has been shown by the measurements of *Hirschberg* that the angle of deviation is about 15° when the image is at the margin of a normally large pupil, and about 45° when the image is at the margin of the cornea of the squinting eye.

Another thing can be determined by this combination of the cover and mirror tests. The prognosis depends in very many cases on whether the strabismic eye still retains the power of central fixation, and this can be determined by covering the fixing eye; if the other eye can fix centrally it will turn at once and fix the little aperture in the mirror, but if it cannot, it will wander about aimlessly. In many cases an excentric portion of the retina is used for fixation.

All of these experiments show that the visual line of the strabismic eye misses the object of fixation. As we have already learned, diplopia ordinarily appears when the visual lines do not intersect at the point of fixation, and all persons with strabismus would therefore be expected to see double, but our patient denies that he has double vision. This brings us to another clinical symptom of strabismus: Although diplopia is the symptom of which patients complain most in the strabismus caused by paresis of the ocular muscles, yet it is usually and characteristically absent in the muscular strabismus with which we are now dealing. This can be readily understood when one eye is very amblyopic, but it is also true when both eyes have useful vision. The reason of this is that the person gives his entire attention to the image seen by the fixing eye and suppresses that of the other. Diplopia is rarely met with in strabismus, and we must employ special test conditions in order to produce it artificially.

For example: we place a red glass before one eye, a green one before the other and direct the patient to look at a light. Under such conditions it frequently happens that he is able to perceive both images at the same time, or, if not, we may alter one image by placing a *Maddox* rod before an eye, covering each eye alternately and then suddenly leaving both eyes uncovered. It must not be thought, however, that the patient can fix his attention equally upon two places, that he can see both the object in the axis of the fixing eye and that in the axis of the other. He sees with the latter only a faint, excentric image of the object toward which his fixing eye is directed. He suppresses the image of the one toward which his strabismic eye is directed and perceives two images of the same object.

We frequently meet with still another phenomenon in the visual act in strabismus, called the abnormal correspondence of the retinæ, by which we understand that if a strabismus exists for a long time certain places on the retinæ, which continually receive images of one and the same object, e.g., the fovea of the good eye and a place situated to the inner side of the fovea of the other, learn to localize their impressions in a common line of sight. Two foveæ do not localize in one and the same direction in such a case, as in a normal pair of eyes, but in different directions, which deviate according to the angle of strabismus. Another place on the retina of the squinting eye has associated itself with the fovea of the other. This anomalous correspondence may persist even after the strabismus has been corrected by operation.

When we have ascertained that a manifest strabismus convergens exists our next duty is to determine exactly the degree of deviation. Various methods for the measurement of the angle of strabismus are at out disposal, but I use almost exclusively the tangent scale of *Maddox*. When the patient fixes the light with one eye, the distance between the light and the point where the visual line of the strabismic eye falls upon the scale is the tangent to the angle of strabismus. The large figures of this scale are used in measuring latent strabismus by ascertaining through what figure the red band of light produced by the *Maddox* rod passes, but we use the small numbers, which give us the length of the tangent at the distance of one metre, to measure the angle of a manifest strabismus.

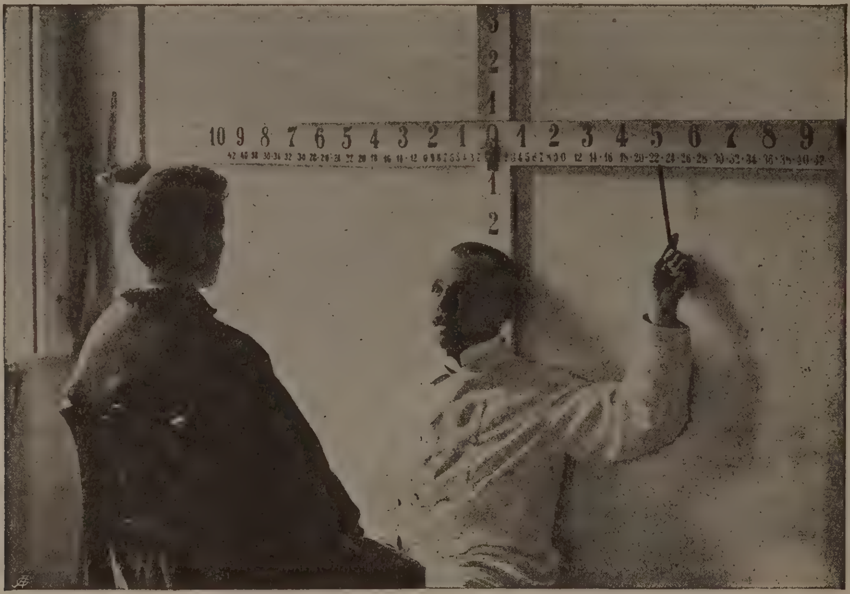


FIG. 111.—Determination of the Angle of Strabismus on the Tangent Scale of *Maddox*.

The patient is placed so that his eyes are on a level with the light situated one metre in front of him. I place myself below the scale, facing the patient, with my head just beneath the light. The patient now looks at the light and I see its reflex image appear on the corneæ of both eyes. The image appears somewhat to the inner side of the centre of the pupil of the fixing right eye, because of the angle γ, and on the outer side of the left eye, between the margins of the cornea and of the pupil. From this we know that the deviation exceeds 15°. I now direct the patient to hold his head still and to look slowly to the left, fixing one number after another as I indicate it with my finger. The reflex image on the squinting eye gradually moves toward the centre of the pupil until finally it reaches the same position that it had occupied on the fixing eye. At the same time I see the displaced image on the first one. This method is very simple. We need only to direct the eyes in any case of strabismus so that the reflex image will occupy the same place on the cornea of the strabismic eye that it formerly occupied on the fixing eye, and then to read off the degree of deviation.

Thus far we have learned that strabismus is present, the amount of the deviation, the vision and the refraction of both eyes. We must now study the movements of the eyes for two reasons.

Every case of strabismus may come from two principal causes. It may be due to paresis of an ocular muscle, of which we will speak more in detail when we come to deal with pareses of those muscles. In such a case the strabismus appears when the eye is moved in the sphere of action of the paretic muscle. For example: if the externus of the right eye is affected, strabismus will appear on looking to the right, for the right eye will not move outward in company with the left. In this patient the strabismus can be seen in whatever way he looks. We bid him look to the right and then to the left, and can perceive at once that these associated movements are perfectly uniform. In fact the angle of strabismus remains exactly the same in concomitant, or muscular strabismus, no matter in what direction the patient turns his eyes.

We will establish this more accurately, for this is the second principal difference between muscular and paralytic strabismus. We determine the angle of strabismus; the deviation of the squinting eye we call the primary deviation. We now cover the fixing eye and compel the other to fix, so that the good eye is in a position of strabismus; this we call the secondary deviation. We test and find that the secondary deviation is exactly the same as the primary. The condition is quite different in paralytic strabismus. The primary position of the eyes in concomitant strabismus is not only not parallel, but crossed.

The other reason why we must test the movements of the eyes is that the choice of the subsequent operation depends on the result.

We close one eye of the patient and have the other make excursions in all directions. The movement of abduction is normal when the margin of the cornea touches the outer commissure; the adduction is normal when the inner margin of the pupil reaches an imaginary line drawn between the two puncta lacrimalia. To test the dynamic convergence I stand in front of the patient, fix with one eye his fixing eye, carry a pencil along the line of fixation toward his eye and so cause him to accommodate more and more. The squinting eye then shows the convergence of both eyes.

Only one thing remains to conclude the clinical examination of a case of convergent strabismus. We have learned what a weighty factor the power of fusion is in the prevention of this condition; we have seen that it is able to hold in check the latent forms, and it is natural to suppose that it is either absent, or insufficient in a case of true strabismus. Such an absence, or insufficient development of the power of fusion is, in fact, the most important cause of this form of strabismus, and therefore we must determine its condition.

The amblyoscope devised by *Worth* is of good service in testing the power of fusion. It consists of two brass tubes united by a hinge and an arc. Each tube is composed of a longer and a shorter one joined at an angle of 120°.

A single mirror at this place reflects the images from translucent picture slides, which are seen through a convex lens. The two halves of the instrument may be adjusted to correspond to a convergence of the visual axes to 60°, and to a divergence of 30°. When we look into the apparatus adjusted for the normal, with both eyes, and pictures are introduced that may be blended, as by a stereoscope, fusion is easy. If we change the angle so as to make the eyes converge, the figures follow and fusion can be maintained up to a certain point.

How is it with our patient, who has alternating strabismus and good vision in each eye? He sees both figures when they are placed wide apart, but as they are drawn together he loses sight of one completely. He does not see them simultaneously when they lie close together, and above all he cannot fuse them. In other cases we find that, after we have adjusted the amblyoscope to the angle of strabismus, the two incomplete figures become fused, but only in a certain position; as soon as we change the angle of the apparatus the images immediately separate. In such a case the patient possesses only a slight power of fusion. This experiment shows that this power may be considerably impaired in any case of convergent strabismus. The investigation of this point is of special importance to the treatment, as well as to the development and etiology.

137. The Etiology of Strabismus Convergens

It is impossible to demonstrate by one or by several cases all of the phenomena we meet with in convergent strabismus, but the following cardinal symptoms are generally to be found more or less marked in every case: 1, The convergence is abnormal; 2, the power of fusion is imperfectly developed; 3, the visual sensation of the strabismic eye is suppressed; 4, its vision is subnormal; 5, usually the eyes are hypermetropic or have hypermetropic astigmatism.

We have now to consider its etiology and how the cause may explain the symptoms. The latent forms of strabismus are caused by mechanical, anatomical factors, that vary greatly in different individuals, which determine the movements of the eyes, and we know that most heterophorias are kept latent and prevented from becoming manifest strabismus by the involuntary effort of fusion. The question therefore arises: Are not the same mechanical factors present, and have they not finally produced the manifest strabismus?

It was formerly believed that convergent strabismus was due either to a considerable shortening of the internal rectus, or to a weakness of the externus, and therefore the only mode of treatment for a long time was tenotomy of the internus.

When the power of abduction is tested in a large number of cases it is found to be normal in about 80%, showing that, in the majority, weakness of the externi and shortening of the interni cannot be the cause. It is only after the strabismus has lasted a long time that a strong spasm develops in the internus. The fact that strabismus frequently disappears when the patient is under general anæsthesia also goes to prove that there is at first no abnormal shortening of the muscle. It also disappears in quite a number of cases when the accommodation has been paralyzed with atropin, a fact that serves as the basis for part of our conservative treatment. We cannot accept this so called muscle theory, but it must not be inferred from this that anatomical anomalies in the position of the eye play no part in the etiology of convergent strabismus. These anatomical incongruencies frequently form a predisposition to manifest as well as to latent strabismus.

Clinical investigations have proved, however, that only very great anomalies in the position of the eyes furnish a direct cause. Slighter degrees of abnormal position may be overcome by the involuntary effort of fusion, when it acts from childhood, and the innervations may be adapted to the needs of binocular single vision. Of what great accomplishments the involuntary effort of fusion is capable we may see from the fact that it sometimes overcomes even greater vertical deviations than we could overcome under ordinary conditions. The innervation is particularly great in such cases. Perhaps among my students, in whom the test for orthophoria reveals a latent deviation of the eyes, may have noticed diplopia when the eyes were tired. The involuntary effort of fusion frequently loses its efficiency, but it causes the diplopia to disappear as soon as the latter is noticed. Mechanical, anatomical errors in the position of the eyes do not suffice alone to cause permanent strabismus. It is only under one condition that a strabismus may develop when the eyes are thus predisposed in consequence of mechanical factors, and that is when the vision of one eye is permanently below a certain limit. When the power of binocular vision has been lost strabismus will appear, at least at times, because of the absence of the movements of fusion, for which binocular vision is a prerequisite. The associated movements of the eyes may appear to be normal when one eye is blind, yet when the seeing eye makes a movement of convergence the blind eye, instead of converging, makes a movement of abduction. Many cases in which the vision of one eye is bad exhibit such a relative, or insufficient convergence when looking at near objects, but fully developed convergent strabismus is not such a relative, but a permanent deviation and if the mechanical factors, on which the movements of our eyes depend, do not suffice for the etiology of this form, something else must be found.

We therefore investigate in how far the dependence of the motor apparatus upon the sensory and dioptric systems must be taken into account. One of the most important clinical symptoms is the fact that the majority of strabismic children are hypermetropic.

We have already discussed the association between accommodation and convergence. As the hypermetrope accommodates more for all distances than the emmetrope or myope a tendency to abnormal convergence is present in all hypermetropes, and *Donders* believed that this tendency to abnormal convergence, produced by hypermetropia and increased accommodation, was the principal cause of strabismus convergens. There is no doubt that it is a very important etiological factor in many cases, but while it is true that most persons with convergent strabismus are hypermetropes, it is equally true that most hypermetropes do not have strabismus. Even among persons who have the highest degrees of hypermetropia a case of permanent convergent strabismus is rare.

The condition of strabismus disappears completely after correction of the hypermetropia with glasses in only a small group of cases. If the inward deviation in hypermetropia were only the consequence of the increased innervation of accommodation we would need only to prescribe the correct glasses, or to do away with the accommodation by means of atropin, in order to cure it, but the strabismus is simply lessened somewhat in many of these children by the glasses, and in many it is not influenced at all.

In the second place hypermetropia cannot be the principal cause of convergent strabismus because many of these patients are not hypermetropic, but emmetropic, or myopic.

In the third place the angle of strabismus is often much greater than would be proportional to the excess of accommodation.

An important factor is still lacking to the satisfactory explanation of the genesis of strabismus convergens. This is the power of fusion. French and, preeminently, English ophthalmologists, *Parinaud*, *Priestley Smith*, *Maddox*, *Worth*, have proved that the essential cause of strabismus is an imperfectly developed power of fusion, and I am convinced that they are correct. The binocular act of vision can relax more or less this connection between accommodation and convergence under normal conditions and so prevent strabismus. The involuntary effort of fusion counterbalances in hypermetropes the excess of convergence by an antagonistic innervation of divergence; it increases the slow convergence in the insufficient accommodation of myopes. These conditions are so regulated from childhood that the accommodation for distance in hypermetropia excites no impulse toward convergence, but is this the case when the involuntary effort of fusion is not present, or is imperfectly developed?

If a hypermetrope, whose power of fusion is absent, or imperfect, looks at an object in the distance he must accommodate because of his hypermetropia, but this impulse to accommodation excites an innervation of convergence on account of the connection between the two. Fusion cannot counteract this innervation of convergence, because there is no desire for binocular single vision, and his visual lines must converge. As such a child wishes to see distinctly he will direct the one of his two eyes which has the better vision at the object. He cannot move one eye without a corresponding movement of the other and therefore a lateral associated movement of the two eyes must take place until one has fixed upon the object; this must cause the other one to be turned more strongly inward, the seeing eye fixes, the other one is strabismic. Thus we obtain a simple explanation of a strabismus convergens when we start from the hypothesis that the most essential factor in its genesis is an absent, or an imperfectly developed, power of fusion. It only remains to be proved that the power of fusion is actually faulty from the beginning in strabismic children. This proof has been established by numerous investigations.

First, there are cases in which the absence of the power of fusion is a congenital defect. Such children develop strabismus early and independently of any error of refraction, they may be emmetropic, or hypermetropic, and they are apt to complain of diplopia if they are operated on later.

The involuntary effort of fusion may be lost gradually during the early years of life. It was present originally, but its development was inhibited and impaired by children's diseases, or other troubles.

Another proof that the absence of the power of fusion is the cause of strabismus convergens is the fact that excellent cosmetic results have been obtained from operation in many cases, and yet the patients have no binocular single vision, they see double and cannot fuse the double images although they lie close together.

When the two eyes point in quite different directions it would naturally be expected that the children should see double, but it is very characteristic of muscular strabismus that they do not in the overwhelming majority of cases. The visual impressions made in the deviating eye are suppressed. This is a purely psychological process, as has already been explained. The permanent exclusion of the images formed in the strabismic eye usually produces an effect, and we find in such eyes a high degree of amblyopia, especially when they have been turned in for a long time.

This child furnishes us an example; he can scarcely count fingers at the distance of three metres with his strabismic eye. Sometimes the amblyopia may be even greater than this. It was formerly believed to be primary and to be the cause of the strabismus, but we know now, from the investigations of *Worth*, and of *Priestley Smith*, that the percentage of amblyopic eyes in these children become greater the longer the strabismus has lasted, and that the degree of amblyopia increases with the duration of the strabismus.

The clinical experience that such eyes are capable of an astonishing improvement proves that the amblyopia is not caused by congenital anatomical lesions. It has been observed repeatedly that strabismic persons who have lost their better eyes, have regained in later years a good vision in their amblyopic ones through practice and use, and we shall learn in the treatment of strabismus that the amblyopia of the squinting eye is a functional disturbance which is capable of improvement. Amblyopia is not the cause, but the result of strabismus.

Congenital amblyopia may be supposed to exist in a small group of cases, in which the vision is not apt to be as bad as in the neglected cases of strabismus convergens.

If we recognize a defect in the power of fusion to be the most important cause of strabismus we can understand how it is that it may occur not only when hypermetropia is present to act as an exciting factor, but in other conditions of the refraction as well. Anisometropia generally is a predisposing factor, and the deviation may become manifest after fevers, excitements, and convulsions.

Our entire conception of strabismus convergens is that it is an abnormal innervation which may be developed from an imperfect power of fusion by many nervous factors. Intermittent strabismus must be ascribed to purely nervous causes beyond a doubt. There are children in whom a latent convergence cannot be detected, sometimes for days or weeks, and then suddenly a manifest strabismus appears, and later disappears. These cases are differentiated from those of periodic strabismus by the fact that in the latter the deviation can always be demonstrated, either as latent or as manifest. Cases are also met with in which the angle of strabismus fluctuates widely without any demonstrable cause.

We cannot say that anatomical anomalies in the position of the eye need not be taken into account etiologically, for we sometimes meet with cases in which the constant deviation is not dependent on the refraction. We also see the strabismus become less at the time of puberty and we may very well imagine that the anatomical position of the eyes may change during the period of growth. We must also admit that secondary anatomical changes may take place in the muscles and their insertions when a convergent strabismus has existed a long time. An increased tonicity of the interni is particularly common. On the whole we will not be in error if we consider strabismus convergens to be a disturbance of innervation. This is indicated also by the clinical experience that the constitutional predisposition to strabismus is so readily transmissible by heredity, and that other abnormal symptoms of neuropathic taint are also met with in these cases.

138. The Conservative Treatment of Strabismus Convergens

It must seem probable from what has been said concerning its etiology that this form of strabismus may be cured without operation by timely and suitable treatment. This is an advance that has been made gradually in ophthalmology during the past ten years.

Still, we cannot do without the operations for strabismus, for we meet with cases in which the conservative treatment was not used at the right time, or in which it has failed, or in which it may fail from the start. The ophthalmologist has therefore to decide during the course of the examination whether to employ conservative or operative treatment.

Inasmuch as everything is finished in no case of strabismus by the operation, but prolonged observation and repeated examinations are needful throughout the treatment, many of the duties in this field fall to the lot of the general practitioner. Therefore I shall first consider what the general practitioner should do when a little child is brought to him, whose parents have noticed the commencement of a squint, and in whom a concomitant convergent strabismus has been diagnosed. He has a very important duty to perform. Many parents of such children, for whom there is no recourse except an operation, tell us that a physician told them years before that the strabismus would pass away of itself, that it would be outgrown, that they must wait, that it was too early for treatment. These are antiquated and crazy theories which cannot be condemned too strongly.

It is never too early for the treatment of a developing strabismus convergens, but it is often too late. Under no circumstances should a physician put off the parents of such children in this manner, for, in the first place, the cases of convergent strabismus that disappear later, with the development of the child and of its ocular functions, are rare exceptions. To be sure we find a diminution of the angle of strabismus as age advances in many cases, but even when the strabismus retrogresses more or less, one thing remains undone, the amblyopia persists. In the second place, the time when the best results can be obtained from conservative treatment is in the earliest years of life.

This is shown best by picturing to ourselves the course of a strabismus convergens which has not been treated. If the vision of the deviating eye was useful at first, amblyopia steadily increases. Many cases of neglected strabismus finally have no central fixation and vision is reduced to the counting of fingers. We must therefore, under certain circumstances, begin the conservative treatment even in infancy. The physician should send children with commencing strabismus to the ophthalmologist, that he may take the initiative, even though they are only a few months old.

What shall we do now for this child, whose convergent strabismus is evident? First, we ascertain the angle of strabismus, then determine the refraction with the skiascope, then learn by means of the mirror test whether the strabismic eye can fix, and then investigate the mobility of the eyes, particularly the abduction and adduction. Then comes the first step of treatment: We instill one or two drops of a 1% solution of atropin in each eye for three successive days. For what purpose? Such children are usually hypermetropic and a portion of their hypermetropia is latent, concealed by the accommodation, but we must know the refraction of such eyes accurately in order to be able to judge what influence the excess of accommodation exerts upon the convergence. A single instillation of atropin is frequently insufficient to completely paralyze the accommodation, and we also need time to win the child's confidence. This has been done in the present case; the angle of strabismus and the refraction are again measured accurately and we are ready to proceed to the second step of treatment, the prescription of the correcting glasses.

Children with strabismus cannot begin to wear the correcting glasses too early. Like *Priestley Smith*, *Worth*, and *Bielschowsky*, I myself have had infants wear glasses with good results. Sensible parents easily overcome the ridiculous objections raised against this measure, and "the gods themselves vainly strive against stupidity."

Another source of anxiety to the parents, that the children will fall, break their glasses and injure their eyes, can be dissipated. Experience has shown that dangers to the eyes do not accrue through the glasses. When children injure their eyes it is usually with quite different objects.

A more important question is how the glasses shall be adjusted. The lenses must be centred for distance and must be as large as possible that the child may not see over them. They must be fastened on when we are dealing with very small children. A matter of greater interest to us is what glasses to order. All astigmatism in excess of the physiological amount must be corrected, together with an average of four fifths of the total hypermetropia. Full correction of the total hypermetropia is to be avoided, because in many of these children a portion will become latent again after the effect of the atropin has passed away, and then the children will see, indistinctly, something we wish to avoid.

If we happen to have a case with myopia a doubt may arise as to whether it is best to correct the error of refraction, because the tendency to convergence will be augmented by the necessarily increased accommodation, but experience has shown that the normal relations between accommodation and convergence should be maintained in such cases also by means of correction with glasses. After the proper glass has been worn for a while the first question in the etiology of strabismus convergens has been answered. If the hypermetropia and the excess of accommodation form the only cause of the strabismus the latter disappears gradually and there is nothing else to do for the child except to have him wear the glasses constantly.

The strabismic eye is often amblyopic and the amblyopia grows worse the longer the strabismus persists. We must therefore try to preserve, or improve the vision of this eye; it must be induced to take part in the act of vision. The amblyopia of such strabismic eyes can be improved to quite a considerable degree in many cases, as has been proved by numerous investigations into the results produced by the conservative treatment of strabismus. Exceptionally good evidence is furnished by cases in which the better eye has been lost later in life by an accident and the patient has had to depend on the amblyopic one, many of which have been reported. I once had under my care a patient whose good eye was lost through panophthalmitis; the other one, which had been strabismic and was very amblyopic, attained within a few months a vision of more than half the normal. An iron energy is necessary for the production of such a result, to overcome gradually such a functional disturbance.

What can we do to induce the strabismic eye to take part in the act of vision in the early stage of convergent strabismus? It has been recommended to bind up the better eye for a while, but I have found, like many other ophthalmologists, that this measure is successful only in those children who are cared for unusually well. If left to themselves they will try in every way to displace the covering so as to uncover the better eye.

When the attempt to exclude the fixing eye from the act of vision by means of a bandage has not succeeded we commence the daily instillation of a drop of a ½% solution of atropin in the fixing eye alone, as soon as the child has become accustomed to the glasses. The accommodation of the better eye is paralyzed by this means, so that the child can no longer see his playthings distinctly with it, but is compelled to use his other one for near vision. The associated movement is stimulated until the strabismic eye is brought into fixation and, unless its vision is altogether too poor, the child will see his toys more clearly with it than with the atropinized one. The vision of such eyes may be improved very much in this way, as has been demonstrated by many ophthalmologists, particularly by *Worth*, *Maddox* and *Bielschowsky*. It may happen, as *Worth* has observed, that the eye which was better at first may become amblyopic from this exclusion from the act of vision, so the eyes should be reexamined at intervals of a few months, when the ophthalmologist and general practitioner can work together efficiently. We determine in the course of this treatment whether the angle of strabismus gradually decreases and the vision of the amblyopic eye increases. The younger the children and the earlier this treatment is begun, the better are the results. The best time is between the third and fifth years, but our results would be better and appear more quickly if we were always able to obviate the principal cause, the absence or insufficient development of the power of fusion.

Unfortunately it is only in rare cases that we are able to take this very important factor sufficiently into account, for researches have shown that the development of the fusion power is normally completed in the sixth year, so if we are to awake and strengthen the faulty power of fusion we are obliged to make the attempt at a time when the children are not mature enough for such efforts. Still, even this factor may be obviated with intelligent children and a good power of fusion established. *Worth* devised his amblyoscope for this purpose, but frequently neither the physician nor the little patient has the great amount of time and patience necessary for such exercises. Well to do children are the only ones that can afford such an apparatus and satisfactory results can be obtained only occasionally with the simple stereoscope. Not much can be expected from the latter if the angle of strabismus is very large.

Finally, the care of the general condition appertains to the conservative treatment of convergent strabismus. Anæmia, scrofulosis, weakness following children's diseases, must be properly treated. This also is the duty of the general practitioner. Every treatment has its limits, so we still have to consider how long this conservative treatment should be maintained. Experience has given us the following rules.

The main point is whether the case is one of monocular strabismus. If, in the course of several months, the angle of strabismus certainly decreases, if the vision of the strabismic eye improves, and especially if signs of the appearance of the power of fusion can be detected, an operation at that time is contraindicated. We have to wait, and it really depends on the size of the angle of strabismus whether we have to discontinue the conservative treatment. If the angle of strabismus is less than 15° we need not decide the question as to the final obviation of the squint before the child is ten years old, for if the involuntary effort of fusion is present it may still be able to overcome so slight a deviation. Even when this power is absent the abnormal convergence may diminish of itself as the body continues to grow, and in addition to this the cosmetic defect is very slight when the angle is less than 15°. When the angle is greater than this and remains stationary for months, or better for years, in monocular convergent strabismus, an operation is needed. We have to operate also in alternating convergent strabismus after a period of observation. The vision of both eyes is usually good in such cases, because each is used alternately for visual purposes, and ordinarily a congenital defect of the power of fusion is present, which cannot be remedied.

139. The Principles of the Operative Treatment of Strabismus Convergens and the Operations

Before I describe the operations for strabismus, it is important that the indications for them should be understood. We have two operative procedures at our disposal: tenotomy of the internal rectus and advancement of the external rectus. When the angle of strabismus is large the two have to be combined.

Tenotomy is the simpler and easier operation and under certain circumstances its effect can be gauged better, but it has a distinct disadvantage. The tendon of the internal rectus is divided at its insertion into the eyeball, the tenotomized muscle retracts somewhat into the orbit and becomes adherent again to the sclera at a point farther back than its original insertion. It is evident that the eye can no longer rotate inward as strongly as before; an artificial paresis of the rectus internus has been produced by the tenotomy. Formerly this operation was liked very much because of its simplicity, it can often be performed on ambulatory cases, but the mishaps that may be observed are the more numerous the longer the time that has elapsed since it was performed. Aside from the fact that the eye protrudes somewhat after a tenotomy of the internal rectus, and that the palpebral fissure becomes somewhat wider, the convergent strabismus becomes divergent in many cases, and the divergence is even more disfiguring cosmetically. For these reasons many ophthalmologists have adopted the operation of advancement of the externus, for which *Landolt* is the preeminent advocate. The tendon of the externus is divided at its insertion, the muscle is shortened more or less, and then reattached to the eyeball closer to the cornea than its original insertion. This operation requires a greater mastery of operative technique and is defective in that its effect cannot be accurately gauged. Both operations have their indications, their advantages and their disadvantages, while it is evident to the impartial clinician that the entire operative treatment of strabismus is and will remain incomplete, because we can influence in this manner only the peripheral motor organs, and can take into account the true nature of strabismus only to a very moderate degree. On the other hand these efforts are not to be discredited, for the removal of the cosmetic disfigurement is a great credit to ophthalmic therapy, especially as this result may be obtained by a suitable operation without harm to the eye.

In order to be able to choose the operation indicated in a particular case two questions need to be answered: First, should a tenotomy of the internal rectus be performed at all in this case, or not? If the reply to this question is in the affirmative, that the operation is justifiable, the second question arises: Which of the two permissible operations, tenotomy or advancement, promises the better result in this case? How can we answer the first question, whether tenotomy of the internus is to be excluded from the first, or not? Simply by testing the adduction and abduction of the eyes, especially of the strabismic one. It is self evident that a tenotomy should be performed in no case on an internus the power of which may be said to be just normal or subnormal, but only when the power of adduction is distinctly plus.

I direct our first patient to make a movement of adduction of the strabismic eye. Under normal conditions the nasal margin of the pupil reaches an imaginary line drawn between the two puncta lacrimalia in the movement of adduc-

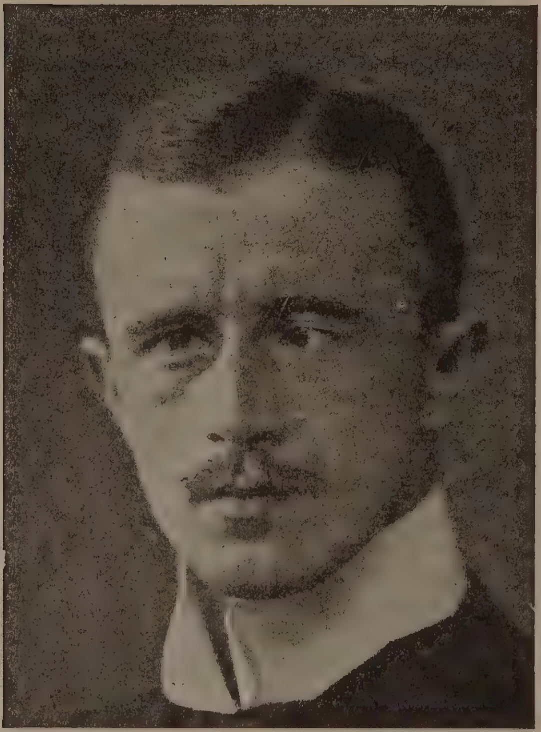


FIG. 112a.—Strabismus Convergens: (*a*) Before the Operation,

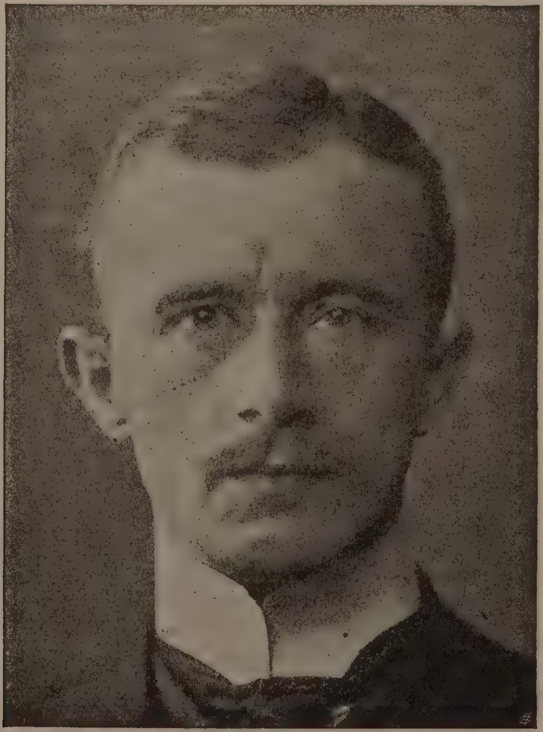


FIG. 112b. (*b*) After the Operation.

tion; if the temporal margin of the pupil reaches to or beyond this line we consider the adduction to be plus.

The abduction also must be taken into account. It is normal in many cases of convergent strabismus, but it may be more or less impaired. We say the abduction is normal when the margin of the cornea touches the outer commissure. When the impairment of the abduction is greater than the increase of the adduction no tenotomy should be performed on the internus. If the result of these examinations shows that tenotomy of the internus is contraindicated, its antagonist may be advanced.

When both operations appear to be permissible the choice depends, first, on the size of the angle of strabismus, and second, on the adduction and abduction. If the angle is about 18°, and the loss of abduction balances the increase of adduction, we tenotomize the internus. If, with this degree of strabismus, the loss of abduction is greater than the increase of adduction, we advance the externus. If the angle is over 20° we have several possibilities at our disposal. We may divide the effect between the two eyes. In the overwhelming majority of cases we choose advancement of one externus, followed some time later by that of the other. This procedure is particularly the one indicated in alternating strabismus. We tenotomize both interni only in exceptional cases, when the patient is an adult with an old strabismus and contracture of these muscles. When the angle of strabismus is still greater, 30° or more, we perform, in monocular strabismus, advancement of the externus and tenotomy of the internus at the same time.

I. Tenotomy of the Internus

Operations for strabismus should be performed under cocain, for we need the assistance the patient can give us, so it is only in cases of necessity, with children that are hard to manage, that we use general anæsthesia. We must bear in mind, in such cases, that the eyes ordinarily diverge during general anæsthesia and that the strabismus disappears to a greater or less degree.

After cocainization of the conjunctiva and introduction of the speculum, we have the patient look toward the side opposite the muscle to be operated on, seize the conjunctiva over the insertion of the tendon with toothed forceps and incise it vertically to the direction of the muscle for a distance of 1 cm. In the same way we cut through the capsule of *Tenon* until the tendon is exposed. We drop in a little more cocain and then follows the measure most important to the successful attainment of the end desired. Aside from the proper indication, everything depends on the production of no overeffect by a tenotomy of the internal rectus. It is self evident that such an overeffect has been produced when a convergent is transformed into a divergent strabismus, but the reduction of the action of the internus below normal must also be considered too great an effect. The operation must not cause any great loss of mobility of the eye inward. If we find in a case on which we have performed tenotomy carefully that the angle of strabismus has not become less, we must allow the wound to heal and then either tenotomize the other internus, or, better, advance one or both externi. We follow the suggestion of *Bielschowsky* in order to avoid an overeffect. We seize the tendon with its fascia and pass a silk thread through it and the superjacent conjunctiva near its insertion, make a little tension on this thread and divide the tendon at its insertion. In this way we have the tenotomized muscle together with the conjunctiva under our control, it cannot retract at random, especially as we are careful to spare as far as possible the connections formed by the fascia between the muscle, the sclera, and the conjunctiva. Then we carry the thread through the anterior margin of the wound, the one nearer the cornea, and tie it loosely. Our patient then sits up and we test the immediate effect of the tenotomy. If the eyes are parallel, or rather divergent, when he is looking straight ahead, the thread must be tightened a little. If the patient happens to be a child under general anæsthesia this test should be made at the first change of dressings, that evening. If the immediate effect is too slight the thread may be loosened, or removed entirely. Both eyes are then bandaged for three days to keep them quiet. During the first days of the healing of the wound we can regulate the effect of a tenotomy by means of this thread. It happens frequently that the effect becomes greater in the course of the first day, when we must try to bring the muscle forward somewhat by traction on the thread, but the final result cannot be known until after two or three weeks. Every other intervention that may be necessary must wait until that time has elapsed.

II. Advancement of the Externus

Very many methods of advancement have been suggested, but the defect is common to all that the immediate effect of the operation retrogrades more or less. It is therefore the rule to try to get quite a little overeffect at first. Various methods have been tried, such as the resection of the tendon, or of the muscle, and the formation of folds, but as long as we do not know in advance how much the immediate effect will retrogress, the result can be gauged very imperfectly whenever advancement is performed alone. I will show in this case a procedure in which I have combined two methods, those of *Worth* and of *Schweigger*. I think it important that the advanced muscle should be attached to the sclera and the sclera to the muscle. We first divide the conjunctiva and *Tenon's* capsule vertically to the direction of the muscle. The wound gapes, but we dissect up the conjunctiva close to the margin of the cornea. Now we introduce a thread through the superficial layers of the sclera near the limbus, parallel to the incision in the conjunctiva and therefore vertical to the direction of the tendon, in such a way that the points of entrance and of exit of the needle are both above the upper margin of the muscle, and pass it in the same way below the level of its lower margin. We have the eyeball firmly under control by means of this thread, which lies visible on the sclera between these two places. Next we place a strabismus hook beneath the tendon, push back the capsule somewhat, and expose its insertion. We replace the hook with *Prince's* advancement forceps, grip the tendon, and the conjunctiva over it, between its arms, and divide the tendon at its place of insertion, together with some other bands of connective tissue. The forceps prevent the muscle from escaping. Now we pass the needles on our first thread from below through the upper and lower margins of the muscle, so that we can tie the ends together later over it and the conjunctiva. The degree of the angle of strabismus determines how far back the needles should be introduced. A powerful effect can be produced in this manner, but it is not yet time to tie the thread. We first introduce two more threads behind the advancement forceps at the upper and lower margins of the muscle, passing through the conjunctiva, capsule and muscle and coming back through the muscle, capsule and conjunctiva. We unite these threads and then have one at the upper and one at the lower margin of the muscle, by means of which we can draw the latter toward the cornea, where we pass them through the superficial layers of the sclera, in a rather divergent direction, and tie them

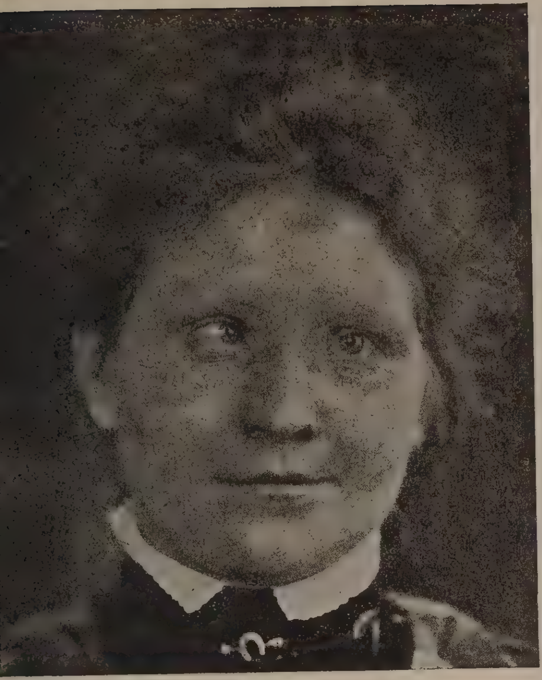


FIG. 113a.

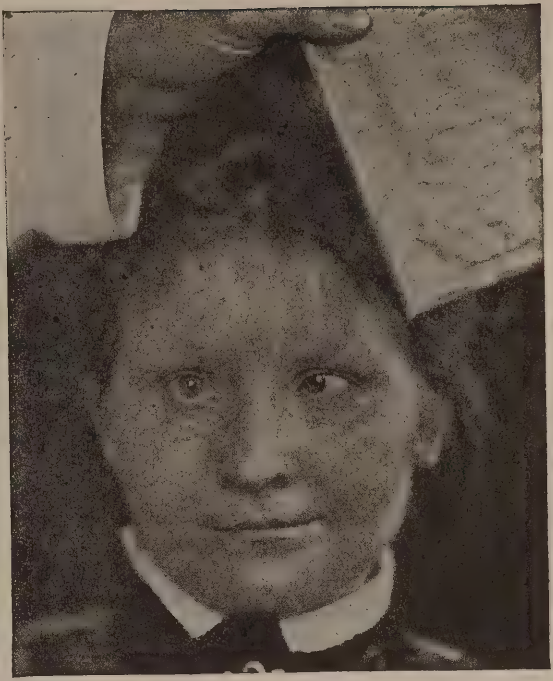


FIG. 113b.

Before the Operation.

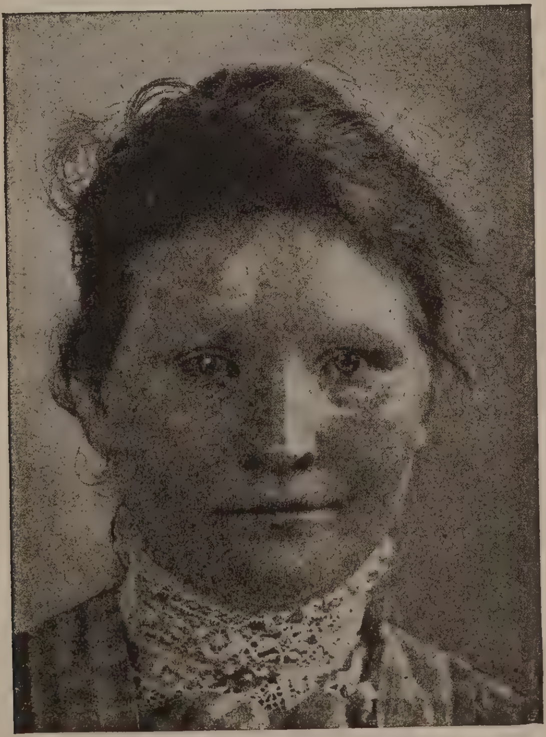


FIG. 113c.—After the Operation.
Strabismus Convergens Alternans with Vertical Deviation.

fast. The insertion of the muscle is brought farther forward in this manner. In order to support it while healing, and to prevent the dissolution of the fresh adhesions during the first few days, we finally tie our first thread over the belly of the muscle and so press it down to a certain degree in its new place of implantation. This last thread must not be drawn very tight, else it will compress the muscle and its nutrient vessels. It may be removed after a few days. The anterior sutures at the margin of the cornea remain in place for eight or ten days. Both eyes are bandaged for from five to seven days. I can recommend this combination of two methods of operation.

III. The Combined Operation

In advancement and simultaneous tenotomy of the antagonist in one eye the technique is the same, but we can gauge the effect better by the combination than by advancement alone. If we wish to rely chiefly on the action of the advanced externus we tenotomize the internus in order to do away with the tension of the antagonist. We do not set back the tenotomized muscle, but tie the thread as soon as the tenotomy has been performed. If the angle of strabismus is so great that the effect of the advancement is insufficient, the internus may be set back and its reinsertion regulated as required by tying the thread loosely.

The treatment of the strabismus is not ended when the patient leaves the hospital; we must try to reestablish binocular vision and to excite fusion. In those cases in which, after the abnormal position has been corrected, there is a possibility of the appearance of the involuntary effort of fusion, the ordinary vocation will suffice to excite it. In other cases, if conditions permit, we prescribe a stereoscope, practice with which teaches the patient to become conscious of the two halves of a picture at the same time. After this has been accomplished the pictures are moved gradually so as to exercise, or utilize, the innervations which counteract the position of, or the tendency to strabismus. The persons who have sufficient patience and energy to do this are certainly rare exceptions. Judgment as to the result depends also on the fact that many patients are troubled more or less by diplopia, after an operation for convergent strabismus, but fortunately this gradually passes away, as a rule.

140. Strabismus Divergens and Its Treatment

The examination of a case of divergent strabismus is conducted exactly in the same manner as that of convergent strabismus. We determine the vision and refraction, measure the angle of strabismus, test the abduction and adduction, ascertain whether the strabismus is monocular or alternating, test for diplopia, and, when possible, investigate the power of fusion. Therefore we need to consider only the etiology and the treatment of this form of strabismus.

We came to the conclusion in the discussion of the etiology of convergent strabismus that it was essentially a disturbance of innervation. Is this the case in divergent strabismus also?

Aside from the fact that the deviation of the eyes is in the opposite direction, divergent strabismus differs from convergent in the following points: It is exceptional for it to develop in the early years of life, but it usually appears when the patients are ten or twelve years old. It is more dependent on mechanical causes. The refraction is myopic in more than half of the cases. The myope needs less accommodation than either the emmetrope or the hypermetrope for near work, and the centre of convergence receives consequently a less powerful impulse of innervation.

Donders supposed that the lack of use of the accommodation led to a weakness of the convergence and then finally to a predominance of the externi over the interni, which resulted in divergence, but this is not a sufficient explanation of the etiology of strabismus divergens, for a total paralysis of convergence may not produce an absolute divergence of the visual axes. A weakness of convergence leads only to a relative divergence, an insufficient convergence for near vision. Two other factors have to be present to produce a real, permanent strabismus.

One is an anatomical predisposition, consisting of a divergent position of the eyes. Such a predisposition may be readily understood to exist in myopia. The fact that the axis of the myopic eye is lengthened explains why many myopes have a latent divergence, an exophoria.

The fusion power must be insufficient to prevent an exophoria from becoming a manifest divergent strabismus. If the power of fusion is insufficient the young myope will complain not only of his nearsightedness, but also that he tires easily and that the lines become confused. Soon he notices that he can read well with one eye if he closes the other, and he can still work after this eye has become divergent. Diplopia does not disturb him, because the excentric image produced in the divergent eye does not excite his weak power of fusion. If the myopia is greater in the divergent eye, its vision is, if possible, less than that of the working eye, so there is no occasion for a disturbing diplopia.

We try to restore the normal relations between accommodation and convergence, in all such cases, by correction of the myopia, or myopic astigmatism. Stereoscopic exercises have little effect on latent or periodic strabismus divergens.

When the power of fusion is wholly absent the diverging eye may be amblyopic, or have a congenital defect of fusion, and in that case operative treatment is the only one to be considered, because the anatomical position cannot be spontaneously corrected. Tenotomy of the externus alone is of almost no avail, for it has been shown that the influence produced by tenotomy of the externus upon the angle of strabismus is much less in divergent, than that of tenotomy of the internus is in convergent strabismus. Clinical observation also teaches us that the immediate effect of such a tenotomy retrogrades very much. A marked overeffect must be striven for in the operative treatment of divergent strabismus. According to *Bielschowsky* a convergence of eight or ten degrees should be obtained immediately after the operation. This can be secured with great certainty by advancement of the internus. We frequently have to combine advancement of the internus with tenotomy of the externus in order to obtain this overeffect when the deviation exceeds ten degrees. When the case is one of monocular strabismus divergens with a high degree of amblyopia of the diverging eye the combined operation is indicated even though the angle of strabismus does not exceed eight or ten degrees.

XIII

The Pupil

141. The Normal Reactions of the Pupil, the Tracts Followed by These Reactions, and the Method of Examination

It is necessary for the physician to take note of the phenomena exhibited by the pupils in many diseases, not only of the eye, but also of the central nervous system, and of the internal organs. It is also necessary that he should be familiar with the normal conditions in order that he may be able to recognize pathological changes when present. For these reasons I shall give a brief review of this subject, and will begin with the examination of the normal pupil.

I. Form and Physiological Size of the Pupil and the Factors by Which They Are Influenced

Normally the pupil is round, but slight deviations from this form may be met with under physiological conditions. Thus it may appear to be not perfectly round in ectropium uveæ, when the pigmentation of its edge is not even.

The physiological size of the pupil, under normal conditions, is from 2.5 to 4.5 mm, but a careful examination will show that it is constantly fluctuating in response to the influence of quite a number of factors, among which are 1, light; 2, sensory stimuli; 3, psychic stimuli; 4, convergence; 5, the closure of the lids. The size of the pupil varies also with the age of the person. It is smaller in infants than in childhood, and does not dilate as much when the light is reduced. It is largest, physiologically, between the fifth and twenty-fifth years, then it gradually decreases in size to about the fiftieth year, and usually becomes still smaller in old age. This phenomenon is due to the increasing rigidity of the walls of the vessels in the iris, as well as to the reduction of the general excitability which takes place as the years advance.

The size of the pupil also depends very much on the condition of adaptation of the eye. When we go from the light into the dark our eyes need time to become adapted to the darkness; in other words, the size of the pupil depends on the relation of the outer brightness to the condition of adaptation of the eye. It is likewise affected by the refraction. Hypermetropes often have small pupils, while myopes, particularly when young, are apt to have strikingly large ones. Sleep has a considerable influence on their size. They are small during natural sleep, and the greatest physiological meiosis is to be met with during deep sleep. The most natural explanation of this fact is that the stimulation which causes the pupils to dilate is absent at such a time.

The pupils dilate in exhaustion, according to *Bumke*. They dilate and lose their reactions immediately after death, but contract again some hours later because of the rigor mortis.

II. Mechanism of the Movements of the Iris

The question arises: By what means is this continuous play of the pupil accomplished?

The iris of the living eye differs from the diaphragm of a photographic camera in that the size of the pupil is regulated automatically. Two muscles are provided for this purpose, the sphincter pupillæ, which makes the pupil smaller, and the dilatator, which makes it larger. In addition, the fullness of the vessels of the iris must be taken into account, for when the amount of blood they contain is increased the iris must become broader and the pupil smaller.

The sphincter pupillæ may be demonstrated histologically with ease, but the existence of a dilatator has been denied, over and over again, and the contention over the mechanics of the movements of the iris has by no means ceased as yet. One thing, however, is certain; a dilatator pupillæ does exist in the iris, as has been proved by *Henle*, *Merkel* and *Koelliker*. It is only recently that it has been possible to make satisfactory histological preparations by bleaching out the annoying pigment from the iris without destroying the structure of its tissue. Considerable differences of opinion still exist in regard to the details. One idea is that the posterior limiting lamella, or *Bruch's* membrane, is muscular. This lamella has been termed the muscular epithelial layer by *Muench*, *Heerfordt* and others. It arises from the connective tissue of the ciliary body, extends forward to the middle of the sphincter, but ends before it reaches the margin of the pupil. *Wolfrum* claims, on the other hand, that the contractive layer of this lamella, which is only 3 or 4 mm thick, is much too thin to be able to supply alone the power by which the iris dilates the pupil. *Muench* calls the network of stroma cells of the iris the dilatator. We will not go any farther into this dispute, for it is not of great importance, and will note simply that in the contraction of the pupil the sphincter has a breadth of 1 mm, and that the central margin is everted with the pigment layer. When the power that dilates the pupil acts, the anterior surface of the iris is made to occupy less than a third of the space previously covered, while its thickness is increased in the proportion of 1:2. Each of the two muscles has its own nerves. The sphincter is supplied, together with the ciliary muscle, by the oculomotor nerve, the fibres of which are conducted to them by way of the ciliary ganglion and the ciliary nerves. The dilatator is supplied by the sympathetic, the pupillary fibres of which come from the ciliospinal centre of the spinal cord.

III. The Physiological Reactions of the Pupil

As the pupil either contracts or dilates it is evident that we have to deal with two principal varieties of pupillary reactions, those of contraction and of dilatation. A feature common to all of them is that they are involuntary and without consciousness.

A. The Reactive Contractions of the Pupil

Contraction of the pupil is produced, 1, by the direct, reflex reaction to light; 2, by the indirect reaction to light; 3, by the reaction of convergence; 4, by the reaction to the closure of the lids; 5, by the reaction to stimulation of the trigeminal and facial nerves.

1. *Direct Reaction to Light*

I must first mention a fact that has been recently established by *Hess*, in order that the reflex reaction of the pupil to light may be understood. Irradiation of every part of the retina will not excite contraction of the pupil, on the contrary *Hess* has proved that only a quite small area about the macula, of about 3 mm radius, is pupillomotor. It was believed formerly that the receptive apparatus was present throughout the retina, but now we have to admit that only this small, central area has any influence on the reflex reaction of the pupil. We cannot irradiate the central, or the excentric portions of the retina alone, because diffused light enters largely into all clinical methods of examination, and it is therefore not so strange that the investigation of the pupils is always faulty in practice. All we can do is to proceed as uniformly as possible. I will illustrate the method of procedure first by irradiation of a healthy eye with a small pupil.

The pupil contracts quickly and then dilates more slowly; then follow some lively little fluctuations and, finally, the margin of the pupil makes incessant minute oscillations, which may be seen very well with *Zeiss'* loupe. This experiment shows at the same time the influence of the adaptation of the retina upon the reaction of the pupil. When we shade the eye first, the entrance of light acts as a stronger stimulus than when the retina has already begun to adapt itself to the light, therefore much smaller quantities of light suffice to excite the reaction of the pupil in an eye adapted to the dark than in one adapted to the light.

When we study the reflex of the pupil to light more accurately we find that a certain time, about 0.4 or 0.5 of a second, elapses between the moment of the entrance of the light and the commencement of the visible contraction. This is known as the period of latency. The entire time occupied by the contraction of the pupil is from ¾ to 1 second.

2. *Consensual Reaction to Light*

Irradiation of one eye produces in man and the higher mammals a contraction of the pupil not only in that eye, but in the other one also. This is known as the indirect, or consensual reaction of the pupil to light. If we irradiate one eye with a certain strength of light the contraction of both pupils is approximately the same, but careful observation has proved that the direct reaction is rather greater than the indirect. I became convinced of this long ago, as have *Bach* and others.

In mammals this indirect pupillary reaction is met with coincidently with the first appearance of the partial decussation of the optic nerves. That we may understand how these reactions take place we will leave our patient for a moment and direct our attention to Plate X, on which is delineated the pupillary tracts and centres.

THE TRACT OF THE REFLEX CONTRACTION OF THE PUPIL

This tract begins in the small, central, pupillomotor area of the retina. The fact that this area alone is pupillomotor destroys the old theory of *Schirmer*, that the light reflex originates in the amacrine, parareticular cells. The rods and cones are to be regarded as the pupillomotor receptive apparatus, according to the researches of *Abelsdorf* and *Hess*. The light stimulus passes from the retina to the optic nerve by which it is conducted along.

The histological researches of *Gudden*, *Key*, *Retzius*, *v. Michel*, and *Bern-*

heimer have proved that the optic nerve contains fibres that differ in thickness. The thinner ones appertain to vision, the thicker ones to the pupil. The latter, in which we are interested just now, pass along the optic nerve to the chiasm, where a partial decussation takes place. From the chiasm they pass along the dorsolateral portion of the optic tract to the corpora quadrigemina, whence the stimulus is transmitted to the nucleus of the oculomotor nerve, by way of which it is returned to the eye. Considerable differences of opinion are still maintained in regard to the details of the course of this tract. All we know is that the pupillary fibres in the optic tract branch off from the corpus geniculatum externum and arrive at the anterior portion of the corpora quadrigemina. According to *Bernheimer* the pupillary fibres may be traced to the region of the nucleus of the oculomotor nerve, while *Dimmer* and *Bach* think that they end in the lateral portion of the anterior corpora quadrigemina. According to *Bach* the fibres that end in the lateral portion of the anterior corpora quadrigemina transmit the stimulus to the cells of the arched and radiating fibres, to which the character of intercalary cells must be ascribed. If this is so, these arched and radiating fibres form the junction with the nucleus of the oculomotor nerve. According to *Held* these arched fibres unite with the oculomotor nucleus, therefore they could transmit the stimulus to the cells of the sphincter pupillæ, which lie in the proximal segment of the nucleus. However this may be we can say that the nucleus of the sphincter is to be considered the starting point of the centrifugal neuron of the light reflex tract of the pupil. The stimulus passes from this place in the trunk of the oculomotor nerve along the base of the skull and into the orbit. The pupillary fibres occupy the inner portion of the nerve. In the orbit they form a part of the branches of the nerve to the ciliary ganglion. We know that destruction of the ciliary ganglion results in absolute immobility of the pupil. The short ciliary nerves arise from the anterior margin of the ciliary ganglion and pass obliquely through the sclera in the vicinity of the optic nerve, run forward in the suprachorioidea, and finally terminate in the ciliary muscle, the iris and the cornea. The course followed by the stimulus, in order to produce a contraction of the pupil, thus appears to be rather complicated.

3. Reaction to Convergence

Whenever our eyes are directed toward a near object the sphincters of both pupils are innervated at the same time as the two internal recti and the ciliary muscle. A contraction of the pupils is produced in this way.

We may test this reaction by having the patient look first at a distance and then suddenly fix on a near object. As the muscle of accommodation acts simultaneously with this reaction of the pupil many researches have been instituted to determine whether the accommodation or the convergence is the active factor in the production of this pupillary phenomenon. At the present time the theory that the contraction of the pupil is associated with the convergence has the preference. Contraction of the internus does not cause contraction of the pupil, but an effort to look at a near object always excites a central impulse which stimulates simultaneously the internus, the sphincter pupillæ and the ciliary muscle.

The tract for this reaction is therefore from the cortex of the brain to the nucleus of the oculomotor nerve, the oculomotor nerve, the ciliary ganglion, the short ciliary nerves.

These three reactions, the direct, consensual, and of convergence, are the most important from a diagnostic point of view, but we have some others that cause the pupil to contract, which should be mentioned.

4. Reaction to the Closure of the Lids

A. v. Graefe observed in 1854 that immobile pupils could contract when the lids were tightly closed. This phenomenon has been studied more recently by *Westphal*, *Piltz* and *Bumke*.

If we hold the lids of this healthy man apart and direct him to close his eyes strongly, the pupils are seen to contract. This is due to an associated movement of the iris, which is stimulated at the same time as the facial branch of the oculomotor nerve. The reaction to the closure of the lids may be both direct and indirect, but the phenomenon has no great diagnostic importance.

5. Reflex from the Trigeminus

A contraction of the pupils can be excited in many persons by stimulation of the trigeminus, aside from the reflex closure of the lids.

6. Ear Reflex of the Pupil

It may also be mentioned that *Schreiber* observed a contraction of the pupil in rabbits, after the optic nerves had been divided, as soon as the root of the ear was seized with force.

B. The Reactive Dilatations of the Pupil

We will turn now to those factors that, by way of innervation, produce a dilatation of the pupil.

1. Dilatation of the Pupil through Stimulation of the Sympathetic

It has been known for a long time that mydriasis is caused by stimulation of the cervical sympathetic. The tract for this reaction is essentially as follows: The fibres that conduct the impulse of dilatation to the pupil leave the spinal cord at the level of the two upper dorsal and the lowest cervical vertebræ. They pass with the anterior branches of the ansa Vieusenii from the ganglion thoracicum supremum to the ganglion cervicale inferius, and from this to the ganglion cervicale supremum through the cervical sympathetic. In this latter ganglion they form a junction with the hypoglossus. After leaving this ganglion they separate from the rami carotidei, which go to join the carotid plexus, and proceed within the skull to the *Gasserian* ganglion, where they unite with the first branch of the trigeminus, with which they reach the eye by way of the long ciliary nerves. These nerves do not pass through the ciliary ganglion.

2. Dilatation of the Pupil by Sensory and Psychic Stimuli

Stimulation of a sensory nerve may excite a dilatation of the pupil. The tract followed is from the sensory nerve to the cerebrum, from the cerebrum to the nucleus of the oculomotor nerve, thence by way of the oculomotor nerve through the ciliary ganglion and the short ciliary nerves to the sphincter pupillæ, the tonicity of which is caused to relax. A psychic stimulus, an impulse of the will, an emotion, and a lively conception may likewise cause the pupil to dilate. The fact is remarkable that the impulse of dilatation of the pupil produced by sensory and psychic stimuli is stronger than the most powerful contraction produced by light. The pupils dilate in anger and terror, and the dilatation continues even when the eye is strongly irradiated. I had the opportunity to study this reaction in a friendly actor while he rendered parts depicting these emotions. The details of the way in which this kind of pupillary dilatation takes place are still uncertain. Some believe it to be an active dilatation, induced by stimulation of the fibres of the sympathetic; others think it passive, due to the relaxation of the tonicity of the sphincter. I think it probable that both factors take part.

METHOD OF TESTING THE REACTION OF THE PUPIL

Bach has stated correctly that we must know four things in order to analyze the most important disturbances of the innervations of the pupil. We must determine its size, 1, with reduced light; 2, with strong irradiation of both eyes; 3, with strong irradiation of only one eye; 4, during convergence.

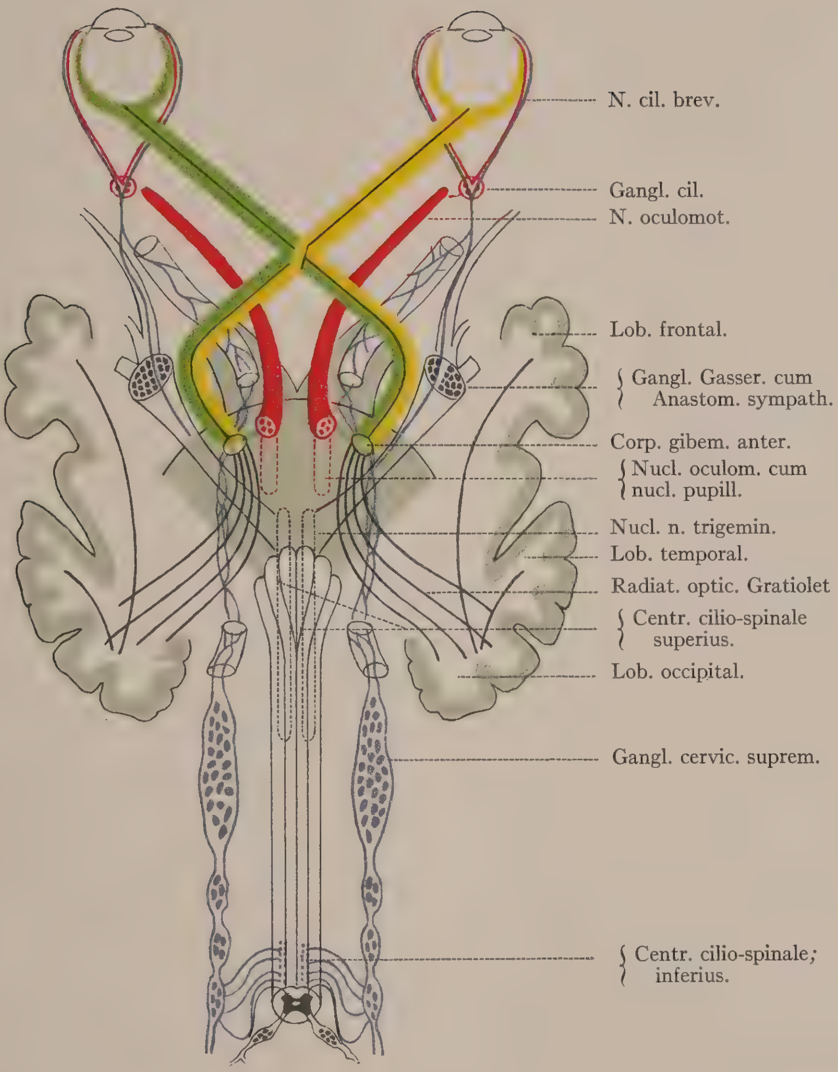
A number of pupillometers have been made, by *Schirmer, Krusius* and others, for the scientific measurement of the size of the pupil, but such instruments are unnecessary for practical work. The examination can be made by such a simple method, that any practitioner can learn to make it in a very short time. The handiest instrument for measuring the size of the pupil is *Haab's* pupillometer, which consists of a strip of white paper, fastened on a ruler, on which the various sizes of the pupil are indicated by a series of graduated black circles differing in diameter by 0.5 mm. This simple instrument is held beside the temple of the patient and the size of the pupil can be determined with sufficient accuracy by comparison.

The test is made in a dark room. A lamp is placed behind the head of the patient, who looks into the distance past, or over the head of the observer, in order that he may exert no movement of convergence, or of accommodation. Light is then thrown into the pupils from a distance of about 40 cm, with the ophthalmoscope; their sizes are compared one with the other and measured by comparison with the pupillometer. In this way we determine the size of the pupils and their reaction in reduced light.

To test the direct and indirect reaction to light we move the lamp forward, close the more distant eye and throw a cone of light into the other one by means of a strong convex lens, after we have first shaded the eye for a little while with the hand that holds the lens. The pupil dilates a little in consequence of this darkening, and the reaction to the light is made more distinct. We ascertain in this way whether the pupil reacts directly to light, and its size is measured again. Now we open the second eye to test the indirect, or consensual reaction, while the other is again irradiated.

Then comes the test for convergence. The lamp is placed once more to the side of the patient, who is suddenly directed to fix his eyes on the forefinger which the examiner holds in front of him.

If the reaction to light is found to be reduced in any case everything depends on whether a slight trace of movement can be detected in the pupil or not. As the central area of the retina is the only portion that is pupillomotor we must cause a strong light to fall on its centre. To do this a lamp may be placed directly in front of the patient, or a bright light may be brought in front of the eye, so that the macula is alternately shaded and illumined, as done by *Bach*. The reaction of the pupils to sensory and psychic stimuli needs to be investigated by the ophthalmologist only in rare cases. Neurologists, on the contrary, need to make these examinations more frequently.



Course of the optic tract.

142. The Most Important Disturbances in the Innervations of the Pupil

We have studied the normal reactions of the pupil and the method of examination. I have now some cases by means of which I can demonstrate the most important forms of its pathological innervation.

Anisocoria in Health

The pupils of this young man are rather unequal in size, but he is otherwise in perfect health.

A great revolution has taken place in our views concerning this condition. Formerly anisocoria was considered almost universally a bad prognostic sign, and I know of several cases in which tabes and progressive paralysis were diagnosed in perfectly healthy people from this symptom alone. We know now that slight degrees of anisocoria occur in healthy persons.

We must ascertain, in every case in which this condition is present, whether it is associated with any disturbances in the reactions of the pupils, for an anisocoria in which such disturbances are not present is harmless. Such inequalities in the size of the pupils may be congenital. When we meet with this condition in a sick person we must remember that diseases of the stomach, lungs, kidneys and liver may cause differences in the size of the pupils, but in such cases we usually find the reaction to light to be perfectly normal. Affections of the sympathetic are apt to be the causes of the anisocoria of internal diseases. When it is improbable that the condition is congenital, and disease of the internal organs has been excluded, the following possible causes must be taken into account: 1, Different refraction of the two eyes; 2, unequal illumination of the two eyes, with preponderance of the direct over the indirect reaction; 3, unlike conditions of adaptation of the two retinæ; 4, inequality of the reaction to the closure of the lids. These four causes are easily dealt with. A difference in the size of the pupils is met with only occasionally when the refraction of the two eyes differs and all other conditions are normal. If the irradiation is not uniform and the direct exceeds the indirect reaction, we need only to make the irradiation uniform and the difference between the pupils will disappear. Anisocoria due to unlike conditions of adaptation may be caused to disappear quickly in the same way, and it is only in extremely rare cases that the reaction to the closure of the lids can be held responsible for a difference in the size of the pupils. The following causes are the ones for which we must particularly examine in practice: 5, A lesion in only one centripetal light reflex tract, or lesions of different degrees in both; 6, a lesion in only one centrifugal tract for contraction of the pupil, or lesions of different degrees in both; 7, a lesion in the tract of the sympathetic, either confined to one side, or present to a different degree on both sides. We will now study these troubles, which are very important in practice. The problems are easier to solve than they appear to be. The disturbances of the light reaction of the pupil that we meet with in practice are, 1, amaurotic immobility; 2, absolute immobility; 3, reflex immobility.

1. Amaurotic Immobility of the Pupil

The right eye of this patient is blind from optic atrophy, caused by a fracture of the base of the skull. The light reflex tract, which begins in the retina and passes through the optic nerve, chiasm, and optic tract until it arrives at the lateral region of the anterior corpora quadrigemina, has been completely interrupted by an injury to the retina and optic nerve. Accordingly we find that when we irradiate the right eye there is no reaction, either direct or indirect, but when we irradiate the left eye the left pupil contracts directly and the right indirectly. The third reaction, that of convergence, is intact. A characteristic of unilateral amaurotic immobility is that the pupil of the blind eye is rather larger than that of the other in reduced light, but the difference in size passes away after the eyes have been uniformly irradiated for a while. In bilateral amaurotic immobility neither pupil reacts to light, either directly or consensually, and both are usually dilated. The diagnosis is easy as a rule: the bilateral reflex immobility of the pupil alone has to be taken into account in the differential diagnosis.

2. *Absolute Immobility of the Pupil*

An absolute immobility of the pupil has been produced in this case by the instillation of atropin. This example will serve to show that absolute immobility is usually due to a deprivation of the sphincter pupillæ of its functional power. It might be thought a priori that it could also be caused by a stimulation of the dilatator, but such a condition can scarcely be demonstrated with certainty in man. In most cases the sphincter is paralyzed. Absolute immobility of the pupil is characterized by absence of the direct and consensual reaction to light, the reaction to convergence, and the dilatation referable to sensory and psychical stimuli.

Various causes may operate to produce such an absolute immobility of the pupil. We must first question whether the condition is one of paresis of the sphincter pupillæ induced by the instillation of a cycloplegic such as atropin, or homatropin, as these drugs are not infrequently used by malingerers for the purpose of deception.

Paresis of the sphincter is often associated with paresis of the muscle of accommodation, forming ophthalmoplegia interna. In such a case we must think of syphilis, but the condition may be produced by other causes, such as ptomaine poisoning from meat, fish or oysters, influenza, diphtheria and lead poisoning. Contusions of the eye very often cause paresis, together with lacerations of the sphincter.

A lesion that gives rise to an absolute immobility of the pupil may be situated in the sphincter, in the short ciliary nerves, in the ciliary ganglion, in the motor roots of the ganglion, in the trunk of the oculomotor nerve, or in the ganglion cells of the nucleus of the sphincter. If the lesion is situated posterior to the ciliary ganglion eserin will continue to act: if in front of it, the effect of the drug will be lessened. An absolute immobility of the pupil may also be produced from the cortex of the brain through inhibition of the tonicity of the sphincter.

When the absolute immobility of the pupil is incomplete, the reaction, called by *Saenger* myotonic, may sometimes be observed. Although the reaction to light has been lost almost entirely, the pupils contract slowly during convergence and remain contracted for some time after the convergence has been relaxed.

3. *Reflex Immobility of the Pupil*

In our next patient, for whom we are indebted to the courtesy of the neurological clinic, we find a reflex immobility of the pupils. There is no direct or indirect reaction to light, and no reaction to sensory and psychic stimuli.

Each eye presents a considerable degree of meiosis. The reaction to convergence, on the contrary, is present and strikingly prompt. These are the characteristic symptoms of a bilateral reflex immobility of the pupils.

This condition may exist in one eye alone and remain thus for a long time. Anisocoria is often present and, as a rule, the disturbance of innervation is farther advanced in the pupil that is the smaller.

When we come to examine the patient more carefully we are struck by a peculiar, fixed expression of the eyes. In many cases the pupils are not quite round, and when the reaction to the closure of the lids is tested we frequently find that the slightest attempt to contract the orbicularis results in a distinct contraction of the pupil. Above all it is characteristic that the reaction to convergence is very prompt.

This derangement of the pupils is of great importance, because it is indicative of tabes and general or progressive paralysis. Its diagnostic significance is frequently of use in the neurology of the eye. It indicates tabes, or taboparalysis, with 90% of probability. These diseases so far exceed all others in which a reflex immobility of the pupil occurs that I mention only for the sake of completeness that a congenital reflex immobility of the pupil has been observed (*Reichardt, Seggel*), that it has been met with in connection with inflammations, tumors and injuries in the medulla oblongata and pons, and that it has been seen after injuries to the head, and even after a contusion of the eyeball. This patient has tabes. If we wish to utilize such a pupillary symptom for purposes of diagnosis we must differentiate sharply between reflex and absolute immobility. The absolute form occurs in progressive paralysis, less often in pure tabes, but in the overwhelming majority of cases the differentiation is made by the test of the reaction to convergence, which is so prompt in the reflex immobility.

In view of this great diagnostic importance of the reflex immobility of the pupil it is a pity that our information is still so insufficient concerning its origin and seat. A very lively interchange of ideas has taken place of late in regard to this subject, but we are still far from definite knowledge. We do not know how to explain the spinal meiosis that is so often associated with it. A diminution of the innervation of the sympathetic may be supposed to take place from a lesion either of the ciliospinal centre, or of the motor fibres in the course of the cervical sympathetic, but then it is strange that we are not apt to meet with the other symptoms which result from a paralysis of the oculopupillary fibres of the cervical sympathetic. It can also be shown that the pupil with spinal meiosis dilates after the instillation of cocain, while such an effect is not produced otherwise in paralysis of these oculopupillary fibres.

Others prefer to ascribe the reflex immobility to a spasm of the sphincter pupillæ, but it is hardly probable that such a condition of motor stimulation would persist for years in tabes, a disease in which we are accustomed to meet with paresis of the other muscles of the eye.

The theory of *Erb* and *Bumke*, that the absence of dilatation of the pupil to sensory and psychic stimuli plays the chief part in the production of the spinal meiosis, seems more plausible. Our knowledge in regard to the anatomical site of the lesion which produces reflex immobility of the pupil is equally uncertain. It has frequently been ascribed to disease of the bundles of *centripetal* pupillary fibres that run from the optic tract to the anterior corpora quadrigemina. According to *Harris* the pupillary fibres partially decussate not only at the chiasm, but also a second time between the anterior corpora quadrigemina and the third nucleus. This second decussation is formed of *Meynert's* fibres and the reflex immobility of the pupil may be caused by a sclerosis of these. *Heddæus* supposes that the ramus iridis of the oculomotorius consists of two twigs, one of which serves for the reaction to light, the other for that of convergence. Numerous other hypotheses have been advanced, of which I will mention only that *Bach* supposes that there are two centres of inhibition for the reflexes of the pupil in the spinal end of the medulla oblongata, one centre to regulate the light reflex by inhibition, the other to regulate the sensory and psychic stimuli to the pupil. Reflex immobility and meiosis may be produced by disturbance of these tracts. The situation of the lesion has naturally been looked for anatomically many times, but it has not been found. It seems to be settled that the roof of the corpora quadrigemina takes no part in the production of reflex immobility of the pupil.

Rieger and *v. Forster* were the first to advance the theory that the site of the lesion was in the cervical portion of the spinal cord. Lesions in the posterior columns have been reported by *Wolff*, *Gaupp*, and *Reichhardt*, which they thought were related to reflex immobility of the pupil, but others, especially *Bumke*, dispute the idea that the site of the lesion is in this place. This important problem remains to be solved in the future. We must be content with the demonstration of these conditions. In amaurotic and absolute immobility the contraction tracts of the pupil are affected; in reflex immobility the dilatation tract is also involved, if we consider the meiosis to be due to a change in the innervation by the sympathetic, or to a deficiency in the response to sensory and psychic stimuli.

A spastic mydriasis, due to stimulation of the fibres of the sympathetic which supply the dilatator pupillæ is of no great consequence. Paralysis of the oculopupillary fibres of the cervical sympathetic is far more common. This creates such an important clinical picture, the so called *Horner's* syndrome, that I shall take pains to demonstrate it as soon as I have an opportunity. At the present time I only wish to say, on account of the connection with derangements of the pupil, that meiosis occurs in that condition because the dilatator pupillæ is paralyzed. The reactions to light and convergence are normal. Weak solutions of cocain do not affect the pupil when the paresis of the sympathetic is complete. In addition to the meiosis the palpebral fissure is narrowed, because the upper lid hangs down somewhat and the lower lid rises a little in consequence of the paresis of the superior and inferior palpebral muscles, which are innervated by the sympathetic. There is also some enophthalmos, due to the paresis of the smooth muscles of the orbit. When the paresis is recent the affected side of the face is the more reddened.

In conclusion I will mention briefly some rare conditions. By hippus iridis is meant a rhythmic contraction and dilatation of the pupil which takes place every few seconds and is usually independent of light, convergence and psychic or sensory stimuli. Such a hippus may occur alone or may be associated with nystagmus; it is observed most frequently in multiple sclerosis, but it is also met with in syphilis of the brain, meningitis, chorea and epilepsy.

Another condition is the so called quickly changing pupils, a phenomenon in which first one and then the other pupil is markedly dilated. This alternate change in the size of the pupils takes place without any demonstrable cause. Sometimes one pupil alone may exhibit such fluctuations in size, which are independent of light and convergence; this has been seen particularly in cases of congenital paresis of the oculomotor nerve, or when such a paresis has developed early in life, when the paretic upper lid may rise and fall periodically in unison with the contraction and dilatation of the pupil.

In former years much was written concerning a paradoxical reaction to light, by which is understood that, instead of contracting, the pupil dilates in response to light and contracts when shaded, but whether such a reaction ever occurs can be proved positively only when the sources of error in the examination of the pupil have been rigorously taken into account.

143. Can a Disease of the Central Nervous System be Diagnosed from the Derangements of the Innervation of the Pupils Alone?

The physician needs to pay much attention to the pupillary phenomena in the domain of internal medicine, of psychiatry, and of neurology, and he may be greatly helped in this respect in the eye clinic. We enter upon a field in which ophthalmology oversteps the limit of her specialty and becomes a coworker in the realm of neurology. In order to give a correct idea of the importance of ophthalmology to neurology I shall demonstrate some cases to show the special importance of the ocular symptoms in the diseases presented. In such cases we have always to consider the question: In how far are the changes in the eye of importance in making positive or completing the neurological diagnosis? On the other hand I can exhibit cases which pass first through our hands, in which we diagnose a disease of the central nervous system from the ocular symptoms and then turn them over to the neurologist for treatment, or for a final diagnosis. In this way the study of the neurology of the eye serves to prepare the student for the study of nervous diseases, which is continually growing more and more important. *The close cooperation of neurology and ophthalmology is an important postulate of medical science.*

It is plain that we should not confine our examination to the eyes alone when a suspicion arises that a disease of the central nervous system is present. We primarily examine the eyes in their entirety; then we see in how far all ocular symptoms are of importance to the diagnosis of the disease of the central nervous system, and we shall learn to recognize the limits of the diagnostic value of these symptoms. We must also take into account the condition of the general organism, so as to group together all of the symptoms in making a neurological diagnosis. Still, the cases are not rare in which the changes in the pupil form the first, or the most marked, morbid symptom. We have such a case in this patient; he came simply because he wished a presbyopic glass. His vision, field and fundus are perfectly normal, but we find in each eye a marked reflex immobility of the pupil and meiosis.

Let me repeat: We speak of reflex immobility of the pupil when the direct and indirect reaction to light and the dilatation caused by sensory and psychical stimuli are absent, and the reaction to convergence is present. In view of such a case as this it is necessary that we should inquire: *In how far can we base, or conjecture, the diagnosis of a disease of the central nervous system from the changes in the movements of the pupils alone?* A priori two ways are open for the solution of this problem. We may ask whether the pupillary symptoms give us any indication of the seat of the disease in the central nervous system. In other words: Does a special phenomenon of the pupil tell us that a focus of disease is situated in this or that place in the central nervous system? Or we may take the individual diseases of the central nervous system and ask: Which of these diseases reveal themselves through derangements in the reactions of the pupil, and which ones can we recognize from these changes? I maintain that the question for us just now is: What diagnosis can we form when pupillary derangements alone are present, as in this case? The first question, whether pupillary symptoms are able to reveal any certain location of a nerve lesion and so guide us to the disease, is quickly answered in the negative. The relations between localization and pupillary symptoms cannot be utilized diagnostically in diseases of the nervous system when the derangement of the innervation of the pupil is the only symptom. No single phenomenon of the pupil alone is able to locate a lesion at a certain place in the central nervous system. We have known for a long time, from numerous experiments made by physiologists and neurologists, that dilatation of the pupils can be produced by stimulation of the brain cortex, and that there are places in the cortex from which the dilatation of the pupil is most easily produced. Such places are the gyrus centralis anterior in dogs and cats, the rhachidian bulb in monkeys, according to *Levinsohn* and *Parsons*. A certain focus is not indicated by this phenomenon alone in man. No single pupillary symptom by itself can indicate a focus in the cerebrum and the intermediate brain. Even in diseases of the intermediate brain, the corpora quadrigemina, the tegmental region and the pineal gland, the behavior of the pupil is very variable. The pupils are influenced in diseases of the peduncle also, but no specific phenomena are produced. The situation is the same with regard to diseases of the pons and cerebellum. The answer and the diagnostic value of the pupillary symptoms are quite different when we come to the second question: What diseases reveal themselves etiologically by derangements in the reactions of the pupils in such a way that we can conjecture or base the diagnosis on the pupillary symptoms alone?

The Pupillary Reactions in the Most Important Diseases of the Central Nervous System

In order to answer this question it is necessary that I should review briefly the behavior of the reactions of the pupil in these diseases, and in order to impress the conditions the more easily I will divide all of the diseases to be taken into account into three large groups: 1, Those in which the pupillary symptoms are so variable and uncertain that alone they furnish no basis for a diagnosis; 2, those in which at least relatively characteristic pupillary symptoms are present; 3, those in which the derangements of the pupil exist as a rule.

I. Diseases in Which the Conditions of the Pupils are not Uniform

This group includes most cases of poisoning, in many of which the pupils are affected, so the reactions should be tested in each case, but the conditions are extremely variable, as may be readily understood. The poison may at first be irritant, and then the irritation may be followed by paresis. The quantity of poison, the specificity of its action, the susceptibility of the patient, and the stage of the poisoning all have to be taken into account. The effect on the pupil may be direct, or it may be indirect and due to the lack of oxygen in the blood caused by dyspnœa or convulsions. It is evident from these facts that the behavior of the pupils must vary a great deal in cases of poisoning.

In acute alcoholism the pupils are often contracted, often dilated; the reactions are sometimes decreased, sometimes increased. In chronic alcoholism the pupils are frequently relatively small and the reactions slow, but this is not always the case. Reflex immobility of the pupil is very rare in aicoholism. The dilatation of the pupil in intoxication is supposed to be due to an irritation of the brain cortex and an inhibition of the tonicity of the oculomotor nerve. The meiosis is explained as a spasm of the sphincter pupillæ.

Meiosis and mydriasis have likewise been observed in nicotine poisoning.

The behavior of the pupil when the patient is under the influence of ether or chloroform depends on the stage and depth of the narcosis. In the stage of excitement the pupils are large and still react to light; when stupor sets in they become small and no longer react. Then, with asphyxia, mydriasis with immobility of the pupil replaces the meiosis; a menacing sign. There is no need that I should enumerate the many other forms of poisoning in which the pupillary symptoms are uncertain; it is better that we should turn to forms of disease in which the pupil exhibits nothing characteristic, with which we may deal briefly. The pupillary symptoms alone are not diagnostic in any of the diffuse diseases of the brain and spinal cord, not even in multiple sclerosis. It is only in syringomyelia that inequalities in the size of the pupils may be observed with comparative frequency, as the result of derangements of the innervation by the sympathetic, while centripetal and centrifugal disturbances of the light reflex tract are not to be expected, because of the anatomical site of the disease. The condition is quite similar when we would estimate alone the diagnostic value of the disturbances of the pupil in the systemic diseases of the spinal cord. One disease only is exceptional, tabes, and in this the pupillary symptoms are extremely important.

We frequently meet with derangements of the innervation of the pupil in the various forms of meningitis. The behavior of the pupil is not typical in epidemic cerebrospinal meningitis; meiosis alternates with mydriasis. In purulent meningitis the variable behavior of the pupils, inequality, fluctuations in size, temporary and then permanent immobility, is likewise characteristic. The pupils are sometimes unequal in tuberculous meningitis; when the disease is in an advanced stage they are usually dilated and not responsive to light. There is nothing characteristic in the behavior of the pupil in diseases of the peripheral nerves.

When we turn to psychoneuroses and mental diseases we find manifold pupillary symptoms in neurasthenia, but they are governed by no fixed law. Restlessness of the pupils seems to be relatively frequent as a sign of the irritability of the nervous system. In hysteria we come across a mutation of theories. It was formerly believed that derangements of the pupil were impossible in hysteria, because the movements of the pupil are not under the control of the will, but meiosis and mydriasis have been observed and many neurologists believe that disturbances of the pupil may be present both during the hysterical attacks and in the intervals between them. The meiosis is ascribed to a spasm of the sphincter, the mydriasis to an inhibition of its tonicity. In the traumatic neuroses the reaction to light may be increased or diminished, and occasionally a marked dilatation of the pupil with prompt reactions has been observed. As anisocoria without disturbance of the reactions may be present normally, it is evident that we can draw no inferences from such symptoms alone. The pupils may not be affected at all in migraine, but sometimes they are. The derangements of the innervation of the pupil have been recognized for a long time in epilepsy, but here again opinions differ. During the fit the pupils are usually dilated and immobile to all pupillary stimuli, and the loss of the reaction to light has been looked upon as a sign of epilepsy in the differentiation between this disease and hysteria, but loss of the reaction to light, and even absolute immobility of the pupils, has been observed in hysteria. When we turn to inhibitions of psychic development and mental diseases we find the reactions of the pupil normal in imbecility and in the overwhelming majority of idiots. It is only in dementia præcox, and its allied diseases, hebephrenia, katatonia, and dementia paranoides, that the absence of the psychoreflexes, the restlessness of the pupils and the reflex dilatation to sensory stimuli are frequent and to some degree characteristic. On the other hand progressive paralysis exhibits very characteristic pupillary symptoms.

II. Diseases with Relatively Characteristic Pupillary Symptoms

In connection with this group I must call attention to a form of poisoning of which I shall have more to say in another place, botulismus. When both pupils of persons who have previously been well become dilated and immobile after the eating of decayed meat, the symptom is so characteristic that we may base the diagnosis primarily upon it. The pupillary reaction is not the only one impaired in such cases, but we find at the same time a paralysis of the accommodation and trouble in swallowing.

Chronic morphinism is another form of poisoning which is often associated with typical changes in the pupils, which are constantly and markedly contracted. Reaction to light and convergence is usually present, but the dilatation to sensory stimuli is reduced. Great care and very much tact must be used to make the diagnosis in this class of cases.

III. Diseases in Which the Derangements of the Pupil are Characteristic

We have now ranged over a large territory in which we have found traces everywhere of changes in the reactions of the pupil, but none of them sufficiently well defined to enable us to diagnose from them with certainty, or even probability, the disease of the organism. We come now to the diseases in which the pupillary symptoms are more characteristic, so that we can often base upon them the diagnosis of the organic disease. Even though all of the diseases that I have enumerated may not be remembered, these three will not be forgotten: syphilis of the brain, tabes and progressive paralysis.

A. Pupillary Symptoms in Syphilis of the Nervous System

The reason why the pupillary symptoms in this disease are so significant is that syphilis shows a certain predilection to attack the region between the pedunculi cerebri, near which lie the centripetal and centrifugal tracts and the nuclei of the internal muscles of the eye. Absolute immobility of the pupil, with or without paralysis of the ciliary muscle, is the symptom we meet with most frequently. This ophthalmoplegia interna affects one eye more often than both. Next in frequency is the amaurotic immobility caused by disease of the optic nerve, of the chiasm, and of the optic tract. This derangement of the pupil is therefore centripetal. Reflex immobility of the pupil is much less common.

B. Pupillary Symptoms in Tabes

Pupillary symptoms are present in over 80% of the cases of tabes. Their special diagnostic value is due to the fact that they may precede the other symptoms of the disease by many years. Reflex immobility of the pupil calls attention to a threatening or existing tabes. A remarkable fact is that this condition may affect one eye alone for a long time. A considerable degree of meiosis and anisocoria are present at the same time in about 40% of the cases. Other pupillary symptoms, such as mydriasis and an imperfect roundness of the pupil, are not typical. Whenever we meet with reflex immobility of the pupil and meiosis we must think primarily of tabes.

C. Pupillary Symptoms in Progressive Paralysis, or General Paresis

Another disease in which the onset is preceded by the same pupillary symptoms is progressive paralysis. Reflex immobility of the pupil ranks first in this disease also. Statistics show that the reaction to light is lost in about one half of the cases. The second most frequent symptom is absolute immobility of the pupils, which are often widely dilated. Anisocoria has also been frequently observed.

These three diseases of the central nervous system are the only ones in which the derangements of the movements of the pupil are sufficiently characteristic to be depended upon when we have to obtain diagnostic data from them alone. This patient has a typical reflex immobility of the pupil; we can exclude every other cause, so we will make a probable diagnosis of tabes and will turn him over to the neurological clinic for a more thorough examination.

It is a matter of great importance that these pupillary symptoms are able to call our attention to these diseases while they are in an early stage, for the sooner proper treatment is instituted the better will be the result, even though it may be that the advance of the disease can only be checked or delayed. This is another reason why I have shown this patient, in whom the derangement of the movements of the pupil alone can be found as yet. It is necessary that the diagnostic value of changes in the pupil should be estimated correctly when they are the only symptoms present. They must be neither overestimated nor underestimated. The more eye symptoms are present the greater is their importance in the neurological diagnosis. Even so our neurological knowledge has narrow limits. In the study of a single organ we must never lose sight of its relations to the entire organism.

144. The Behavior of the Pupil in Diseases of the Eye and the Action of Mydriatics and Meiotics

We need to be acquainted with the phenomena presented by the pupil not only in diseases of the central nervous system, but above all in diseases of the eye itself, and we have to influence the movements of the pupil therapeutically very often. We use drugs, some of which dilate, some of which contract the pupil, and the physician should be accurately informed in regard to their actions. We will consider first how the reactions of the pupil are affected in diseases of the eye.

I. Behavior of the Pupil in Diseases of the Eye

Diseases of the eyelids cause no direct pupillary symptoms.

The infectious forms of conjunctivitis occasionally affect the pupil. I have here a case of pneumococcal conjunctivitis in which the pupil exhibits a distinct tendency to contract, although the cornea is not involved. This contraction is due to an augmented hyperæmia of the iris and a slight iritis caused by the diffusion of toxic products through the cornea and the irritation excited by them in the iris.

Derangements in the movements and form of the pupil are much more common in diseases of the cornea. When the cornea is perforated, either by a wound or by an ulcer, the pupil becomes distorted in consequence of a prolapse of the iris, which produces either a leucoma adhærens, or an anterior synechia. When the cornea has not been perforated the form and reactions of the pupil remain normal, even when the most extensive cicatrizations are taking place, provided that the preceding inflammation has not left changes in the iris. On the contrary almost all fresh inflammations in the cornea affect the size of the pupil and the movements of the iris, but these pupillary changes are always to be ascribed to a secondary involvement of the iris in the inflammation. I need only cite as an example the ulcus corneæ serpens, which is accompanied by an iritis, as a rule. A feature common to all these inflammatory involvements of the iris in diseases of the cornea is the tendency of the pupil to contract. This can be seen in both of these cases, one of which is a parenchymatous keratitis due to hereditary syphilis, the other an obstinate herpes corneæ. Examples of this nature may be multiplied as much as may be desired.

The pupillary phenomena are manifold in diseases of the iris. The reactions are usually normal in the congenital anomalies, such as coloboma of the iris, and our attention is naturally attracted by the change in the form of the pupil.

The sphincter is frequently paralyzed in contusions; in such cases the pupil is dilated and exhibits various degrees of absolute immobility. When the sphincter is torn the form of the pupil may be changed. I can confirm the statement of *Krusius* that if we instill a meiotic in such an eye a meiosis will be produced, but it will frequently be less in degree than that in the uninjured eye.

The pupil exhibits a strong tendency to contract in the overwhelming majority of fresh cases of inflammation of the iris, whatever the nature of the inflammation may be. This contraction of the pupil is one of the symptoms that differentiates iritis from glaucoma. I cannot repeat this statement too often. The tension becomes increased in some forms of iritis, as in iritis serosa, and in such cases we have to take note of the other symptoms, like the deposits on *Descemet's* membrane. When the iris is inflamed it is natural that the reactions of the pupil should be slow and reduced, though still demonstrable. The reactions of the pupil are found to be present after the iritis has passed away and left only individual synechiæ, but when a large number of posterior synechiæ interfere seriously with the freedom of movement of the margin of the pupil the reactions suffer, and they are completely lost when a circular posterior synechia has been formed, or the surfaces of the iris and anterior capsule have become adherent.

The reactions of the pupil are well marked after iridectomy, in spite of the artificial coloboma, as long as the iris is not bound down by adhesions, as for example, to a secondary cataract. Small tumors of the iris do not affect the reactions of the pupil, at least for a long time.

Next to diseases of the iris come those of the ciliary body. Every cyclitis is complicated more or less by iritis, and consequently the pupillary symptoms are the same, except that in serous cyclitis the tendency of the pupil is to dilate, frequently because of glaucomatous conditions.

This brings us to glaucoma, of which I am able to demonstrate again a case of the absolute form. The pupil is dilated, oval, and wholly destitute of reaction. In the first stage of primary inflammatory glaucoma the increased tension and the displacement of the periphery of the iris into the sinus of the anterior chamber cause the dilatation of the pupil, the paresis of the ciliary nerves the slowness of the reactions. In the final stage of the disease, such as is exhibited in our patient, the tissue of the iris is atrophic. In addition there is an atrophy of the optic nerve, which is jointly responsible for the pupillary troubles in all forms of glaucoma, including glaucoma simplex.

Lenticular opacities exert, as a rule, no influence on the movements of the pupils. Even when the lens is perfectly opaque, as in senile cataract, the reactions of the pupil are perfectly normal. This is a symptom to which we must pay heed when we have to decide whether the extraction of a cataract will prove successful or not. It is only when the lens swells greatly, as in traumatic cataract, that disturbances in the pupillary movements may be noticeable. Inflammatory adhesions may also be formed between the iris and a secondary cataract, as in this patient, in whom the margin of the pupil is adherent in several places.

I need only mention the fact that the form and size of the pupil may be affected by luxations of the lens. When the luxation is into the anterior chamber the pupil is dilated very widely.

Diseases of the vitreous have no effect on the movements of the pupil, as a rule. The reactions are normal even when the opacities are very great, but as soon as we have to deal with severe intraocular inflammations, in which other membranes are involved, pupillary troubles appear.

Disturbances of the pupil are much more common in diseases of the retina. In cases of extensive detachment of the latter the pupil is often somewhat dilated, while the reaction to light is less prompt and full, although sometimes the reaction to light may be very prompt in spite of a very great loss of vision. The condition of this reaction evidently depends on whether the central, pupillomotor area of the retina is still well preserved, and still has sufficient nutrition. No great pupillary troubles are met with in any of the other morbid changes of the retina, whether inflammatory, vascular, or degenerative, until the membrane undergoes secondary atrophy. The retinal elements are destroyed by such tumors as glioma, which explains the derangement of the reaction to light that they cause. The reaction is also affected later by the increase of tension, so that it often happens in glioma that the pupil is dilated and not responsive to light.

Disease of the optic nerve is the principal source of pupillary disturbances. We find an enormous dilatation of the pupil and a reduction of its reaction to light even in the early stage of a choked disc. It is still uncertain how these troubles are brought about; it may be that they are due to disturbances in both the centripetal and the centrifugal conduction in some cases, while in others the loss of vision and the impairment of the reaction to light do not go hand in hand. The latter may be almost completely lost even when the vision is still good. The conditions are quite analogous in other inflammations of the optic nerve. Reduction of the vision and of the reaction of the pupil to light may correspond, but they need not. Even in retrobulbar neuritis, in which an affection of the papillomacular bundle is present, the reaction of the pupil to light may still be prompt in spite of a great impairment of the central vision. It is evident that the visual fibres and the pupillary fibres are not equally affected in such a case.

The pupil behaves in a similar manner in optic atrophy. The loss of vision and of the reaction to light may not be proportional. Thus, *Reichhardt* found prompt reaction to light in spite of amaurosis in a case of monocular blindness that presented the ophthalmoscopic picture of optic atrophy. The anatomical examination revealed the fact that great numbers of the slender visual fibres in the optic nerve were degenerated, while the thick fibres were still normal. Such cases as this are exceptional, however. Generally speaking, we may say that the reaction of the pupil to light is absent in total atrophy of the optic nerve, and that the pupils themselves are dilated, as a rule. Inversely, the presence of the reaction to light tells us that the eye has perception of light and, when we are engaged in the examination of persons suspected of aggravation or simulation, we are justified in doubting the statements that the vision is bad when the reaction is normal and we can find no objective condition in the eye to explain the loss of vision. In total optic atrophy of one eye the pupil is somewhat larger than the other in moderate degrees of light. Reaction to convergence and the dilatation of psychic stimuli are both present.

There is little to add with regard to other diseases in the vicinity of the eye. The optic nerve, as well as the long and short ciliary nerves, may be affected by diseases, inflammations, or tumors in the orbit. The dilatator and sphincter pupillæ may be stimulated or paralyzed, in the same way, or the centripetal conduction of the optic nerve may be interrupted. In injuries of the skull hemorrhages may take place into the orbit and into the sheath spaces of the optic nerve, and impair in this way the reaction to light.

II. Mydriatics and Meiotics

The physician must not only observe the phenomena exhibited by the pupils in disease of the eye, he needs very often to influence them therapeutically. Poisons that act on the movements of the iris are used for this purpose; those that dilate the pupil are called mydriatics, those that cause contraction are named meiotics. It is well for students to have these drugs instilled into their own eyes, as their personal experiences will be best remembered.

The most important mydriatic is atropin, which we use in the form of the sulphate in a 1% solution. If a drop of this solution is instilled into the conjunctival sac the pupil begins to dilate in about ten minutes, and the full effect is obtained in from twenty to thirty minutes. The pupil is then widely dilated, absolutely immobile, and the ciliary muscle is paralyzed at the same time. This mydriasis and paresis of the accommodation lasts for a considerable time; it begins to pass away on the third or fourth day, but the effect of the drug may sometimes be still present at the end of eight or ten days. We have to take into account various individual conditions when dealing with a healthy eye. The pupil of an infant does not respond as promptly to atropin as does that of an older child, according to my observation, and it is a well known fact that the pupil cannot be dilated in this way as easily in old as in young persons. In addition to this, the absorption of the atropin into the eye varies a great deal, and depends, among other things, on the strength of the solution.

How is the effect of atropin produced? It cannot be that the drug is absorbed into the circulation and acts from the blood, for in that case both pupils would become dilated, while in fact the only one affected is the pupil of the eye into which the drop has been instilled. The effect of all of our mydriatics is local.

Atropin passes by diffusion through the cornea into the aqueous. This may be proved by a simple experiment. If a drop of the solution is placed in the conjunctival sac of an animal, the aqueous withdrawn a little later with a syringe and some of this aqueous is instilled into the eye of a second animal, the pupil of the latter dilates. The atropin in the aqueous acts upon the iris, and it has been proved by *P. Schultz* that it paralyzes the terminal filaments of the short ciliary nerves in the sphincter itself. This explains how it is that the pupil can be dilated still more widely in atropin mydriasis by stimulation of the sympathetic. We need atropin above all in iritis, in order to counteract the hyperæmia of the iris, the contraction of the pupil, and the formation of posterior synechiæ, but two things must be borne in mind in practice: One is that the duration of the effect of the drug is very long in the normal eye, so it must not be used to dilate the pupil for the sole purpose of diagnosis. Patients are justified in protesting against such treatment, as we have other means at our disposal with which this purpose may be accomplished.

We must be very careful about the use of atropin in old people, because when they are predisposed to glaucoma the prolonged dilatation of the pupil may excite an attack. Cases have been reported in which a single drop of atropin has excited an attack of fulminating glaucoma which resulted in irreparable blindness. Although such cases are rare it behooves us to be careful in the administration of atropin to old people. If an attack of glaucoma happens to be excited eserin must be instilled several times a day for a number of days.

The second thing we must remember is that general toxic symptoms frequently appear after a prolonged use of atropin, because a portion of the poison escapes from the conjunctival sac into the nose and is absorbed. This intoxication is particularly apt to be produced when the drug is placed in solid form on the mucous membrane in order to obtain a more powerful effect. The symptoms are dryness of the throat, redness of the face, acceleration of the pulse, a general weakness and sometimes loss of consciousness. Discontinuance of the atropin and a subcutaneous injection of morphin as an antidote obviate these troubles.

Substitutes for atropin are daturin, duboisin (hyoscyamin) hydrochlorate, and hyoscin hydroiodate (½%), but the last is still more poisonous than atropin. Scopolamin hydrobromide (1/10%) is another substitute.

I recommend the use of homatropin to dilate the pupil for diagnostic purposes. It requires more time, as much as twenty minutes to dilate the pupil, but its effect passes off more rapidly and has disappeared by the next morning, as a rule. Euphthalmin hydrochloride, in a 5% solution, has a similar effect. This group of mydriatics contrasts with another which produces a dilatation of the pupil through stimulation of the sympathetic, the principal one of which is cocain.

Cocain hydrochloride in a 1% or 2% solution dilates the pupil through stimulation of the terminal filaments of the cervical sympathetic in the eye, as is proved by the fact that if the fibres coming from the superior cervical ganglion are divided these weak solutions of cocain are no longer able to dilate the pupil. It is an old saying that cocain stimulates the dilatator, while atropin paralyzes the sphincter pupillæ. This explains why it is that we instill both drugs in order to obtain the greatest mydriasis. The mydriatic effect of a weak solution of cocain appears in from five to ten minutes. It is not extreme and the reactions to light and convergence are preserved, so this drug is to be recommended as a means for dilating the pupil for diagnostic purposes, and it is of good service in ophthalmoscopy.

Several other things are to be seen when cocain is instilled into the eye. In a short time the conjunctiva becomes pale and has a peculiar feeling of numbness or of cold. Reflex winking ceases because of the loss of sensation, so we have to be careful that the surface of the cornea does not become dry whenever an eye is cocainized. The best way to accomplish this is by covering the eye with cotton wet with boric acid solution. The palpebral fissure becomes a little wider, a slight degree of exophthalmos may appear, and the intraocular tension may be slightly reduced. All of these symptoms are due to the fact that cocain stimulates the fibres of the sympathetic. The vessels are contracted so that the conjunctiva becomes pale, and the muscles in the lid supplied by the sympathetic, the tarsalis superior and inferior, are caused to contract and so increase the size of the palpebral fissure. Most marked of all is the anæsthesia, in which the contraction of the vessels cooperates.

The extracts of the suprarenal capsule, adrenalin, suprarenin, atrabilin, etc., likewise dilate the pupil through stimulation of the dilatator pupillæ, but this stimulation is not transmitted by way of the ciliary nerve, as is the case with cocain. These preparations act rather on the muscle cells of the dilatator, or on the appertaining ganglion cells, for the effect they produce remains exactly the same after the ganglion supremum has been extirpated.

Meiotics

The action of meiotics is the exact opposite of that of mydriatics. The two drugs are physostigmin or eserin salicyate, in solutions varying from ½ to 1%, and pilocarpin muriate in a 1% solution.

The effect of eserin may be noticed a few minutes after a drop has been instilled. The pupil begins to become smaller, because the drug excites the sphincter to contract. The pupil is extremely small in from twenty to thirty minutes, when the effect has reached its maximum. The ciliary body contracts together with the sphincter and its spasmodic contractions may be traced as soon as an attempt is made to fix on a near object. These contractions are sometimes painful. Naturally the eye is adjusted for the near point. Pilocarpin acts in a similar manner, though not so strongly. A meiotic that is seldom used is known as arecolin (1%). The effect produced by meiotics does not last as long as that of atropin, but has usually passed away at the end of a single day.

XIV

Pareses of the Ocular Muscles

145. The Normal Functions of the Extrinsic Ocular Muscles and the Cardinal Symptoms of Paresis of One of Them

When a middle aged or elderly person complains that he has suddenly begun to see double, we are justified in a suspicion that he has a paresis of one of the muscles of the eye, and we need only to direct the patient to close one eye and then the other alternately in order to practically confirm or disprove this diagnosis without any other kind of an examination. The diplopia disappears when one eye is closed, because it is binocular and due to the loss of equilibrium between the eyes in paresis of the ocular muscles. We may readily convince ourselves that such a loss of balance may cause diplopia by looking at a light and pressing a finger against the side of one eye.

Monocular diplopia, met with after injuries, in iridodialysis, in luxations of the lens, and also in incipient cataract, is due to the fact that the two images of the same object are cast upon the one retina and is differentiated from the diplopia caused by paresis of the muscles by its persistence when the other eye is excluded from the act of vision.

So far the diagnosis is very simple, but we must make a much more careful examination of every case, for such pareses are of interest to the physician on account of their great importance in the neurological diagnosis, rather than because of these subjective troubles.

The first question is: Which muscle is affected? To answer this we need to review briefly the actions of the muscles of the eye and to refresh our memory in regard to some anatomical and physiological facts.

Each eye is acted upon by six muscles: 1, The rectus externus; 2, the rectus internus; 3, the rectus superior; 4, the rectus inferior; 5, the obliquus superior; 6, the obliquus inferior. All of these, with the exception of the obliquus inferior, arise from the tendinous ring that surrounds the optic foramen. These muscles diverge as they pass forward and form a funnel which contains, in addition to the orbital fat, the optic nerve, together with the other nerves and vessels that supply the eye. The rectus externus is innervated by the abducens, the obliquus superior by the trochlearis, all of the others by the oculomotorius.

The action of the individual muscles may be demonstrated on a manikin. That of the internal and external recti is the simplest. The external rectus runs parallel to the horizontal meridian of the eyeball, is inserted into the sclera about 7 mm from the temporal margin of the cornea, and abducts the visual line. The internal rectus follows an analogous course on the inner side of the eyeball, is inserted about 5 or 6 mm from the margin of the cornea, and adducts the visual line.

The course and action of the superior rectus is different. When the eyeball is directed straight forward this muscle forms an angle of about 23° with the visual line, so it can elevate the latter without producing any other effect only when the eye is abducted this number of degrees. When the visual line is directed straight forward the eye is rotated inward at the same time that it is elevated by the muscle. The more the eye is adducted the greater is the inward rotation, or the more the inclination inward of the vertical meridian is increased, while the elevation produced is correspondingly decreased. The adducting power of the muscle is slight.

The course of the inferior rectus is quite similar to that of its antagonist, the superior. It lowers the eye, but this depression is associated with no other effect only during an abduction of 23°. It rotates the vertical meridian outward, and this component of its action becomes greater the more the visual line is adducted, i.e., the larger the angle that is formed between the muscle and the visual line. In addition to this the inferior rectus exerts a slight power of adduction, the same as the superior.

The superior oblique passes along the median wall of the orbit to the trochlea, where it describes an angle of 50° and passes laterally backward and somewhat downward to be inserted beneath the superior rectus behind the equator of the globe. We need to take into account only the portion lying between the trochlea and the insertion in the study of the action of this muscle. When the eye is in the primary position it depresses and abducts the visual line. When the latter is abducted until it is vertical to the line of action of the muscle, the superior oblique can only rotate the upper pole of the eye inward, but when it is strongly adducted the sole effect that can be produced is a depression of the visual line.

The inferior oblique is the only muscle that does not arise from the origin common to all the others. It arises anteriorly from the lower wall of the orbit and passes over the inferior rectus backward, outward and upward. It lifts the visual line outward and upward from the primary position, and inclines the upper pole of the eye outward toward the temple. The lifting component is stronger than the rotating one when the visual line is adducted, while the opposite is true during abduction. So much concerning the *individual actions* of the muscles.

We have yet to consider two questions before we return to the examination of our patient. 1, How do these muscles act in movements of the eye? 2, How do the muscles work together upon both eyes?

In regard to the first question, we have only the external and internal recti to turn the eye to the right or the left, while two muscles act together to raise or lower it. The elevators are the superior rectus and the inferior oblique; the depressors are the inferior rectus and the superior oblique. The teleological importance of the associated action of the elevators and depressors is that we can raise and lower our eyes over the greatest part of the field of fixation, for at the moment when the action of one elevator or depressor upon the vertical position of the visual line is relaxed, during the lateral movements of the eyes, the effect produced by the other elevator and depressor is increased. In rotation of the eyes, on the contrary, these elevators and depressors, which work together, are antagonists. The superior rectus and the superior oblique rotate the vertical meridian inward, while the inferior rectus and the inferior oblique rotate it outward. When the muscular balance is normal, this meridian remains perfectly

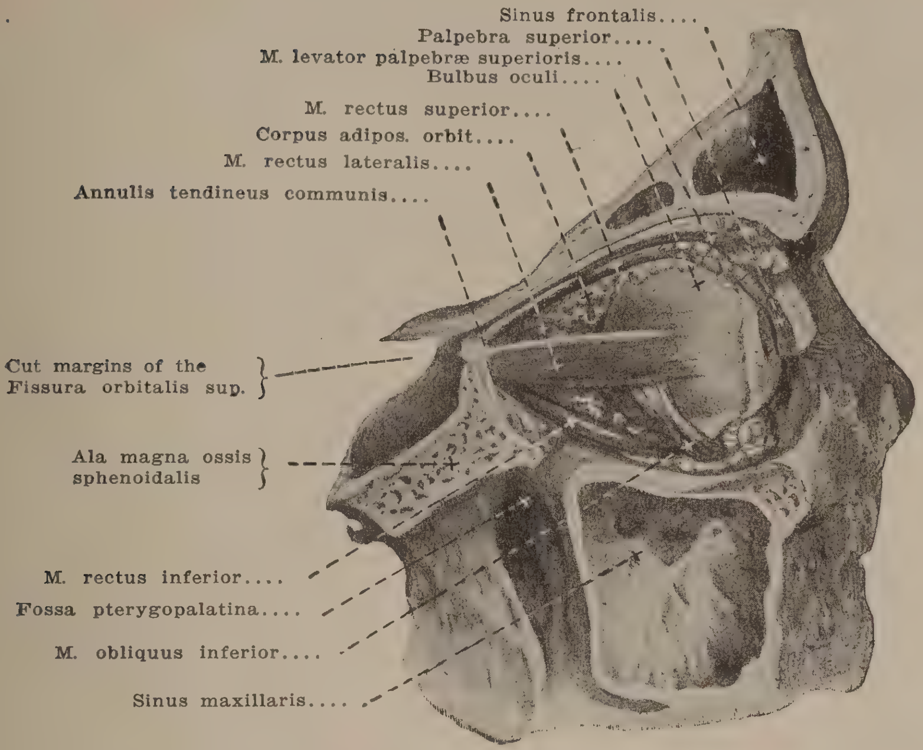


Fig. 114.—Right Orbit Seen from the Right Side (after *Spalteholz*).

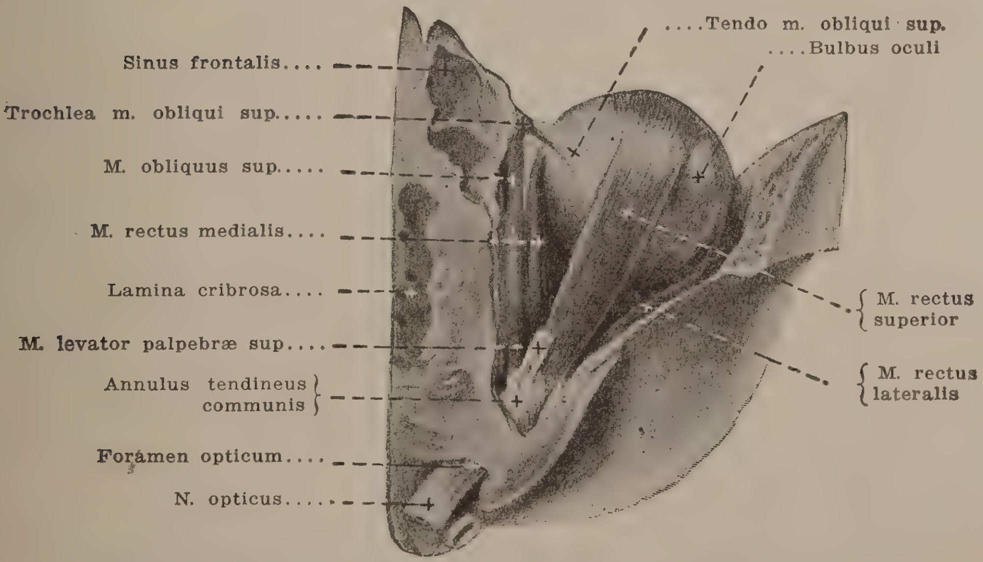


Fig. 115.—Muscles of the Right Eye Seen from Above (after *Spalteholz*).

vertical when the eye is raised or lowered, as well as during purely lateral movements.

All the muscles try to draw the eye somewhat backward, but this traction is counteracted in part by *Tenon's* fascia, from which, in the region of the equator, numerous tags extend to the walls of the orbit and to the lids.

How do the muscles of the two eyes work together? By means of the law that both eyes are always innervated at the same time. If we turn our eyes to the right an equal impulse is communicated to the right externus and the left internus; if we look to the left the left externus and the right internus work together. The tonicity of the acting muscles is increased, while that of their antagonists is relaxed. If we make a movement of convergence the two interni are uniformly innervated, while the tension of the externi is relaxed. Our visual lines may also assume a rather divergent position, as has been shown in the determination of the negative convergence. All of the muscles are slightly tense when we are looking straight ahead.

As regards the field of excursion, the visual line can be adducted and abducted from 43° to 50° under normal conditions. Depression of the eye is rather greater, from 50° to 60°, while its elevation is much less, between 20° and 50°, depending on the relative position of the eyeball to the orbit. We call the entire space in which we can fix an object with both eyes the binocular field of fixation.

Now we can return to our patient and to our first question: Which muscle is paretic? When a muscle is paretic the eye to which it belongs must naturally be retarded in its movements when called upon to move in its sphere of action. Hence, in order to ascertain whether a muscle is in this condition we have the patient keep his eyes fixed on an object, like a pencil, that is moved about in all directions, while we watch to see if we can detect any lagging behind on the part of one of them as they follow these movements. In this case the right eye is quickly seen to be very backward in abduction, so it is evident at once that the external rectus of the right eye is paretic.

This is the *first symptom of paresis of an ocular muscle, an impairment of mobility.*

Another symptom has been brought out by this test. When the eyes are looking directly forward the right one does not seem to be directed exactly at the object of fixation, but to be deviated inward. This deviation of the affected eye is due to the fact that the tonicity of its antagonist overbalances that of the weak muscle; in this case the internal rectus overcomes the external rectus and draws the eye inward a little. This gives us the *second symptom of paresis of the ocular muscles, the paretic strabismus.*

We must study this paretic strabismus rather carefully, for it differs in important particulars from muscular or concomitant strabismus. If we have our patient fix his eyes on a light placed in front of him the positions of the reflections can be seen to be asymmetrical. The reflection on the left eye is a little to the median side of the centre of the pupil, on the right eye it lies to the outer side. The visual line of the right eye therefore passes to the inner side of the point of fixation and the eye deviates inward, because, as I said, the internus preponderates over the paretic externus.

We call the deviation of the paretic eye the primary deviation. When the patient looks toward the right the strabismus becomes more and more distinct, because the farther the eye should move in the sphere of the paretic muscle the more it lags behind.

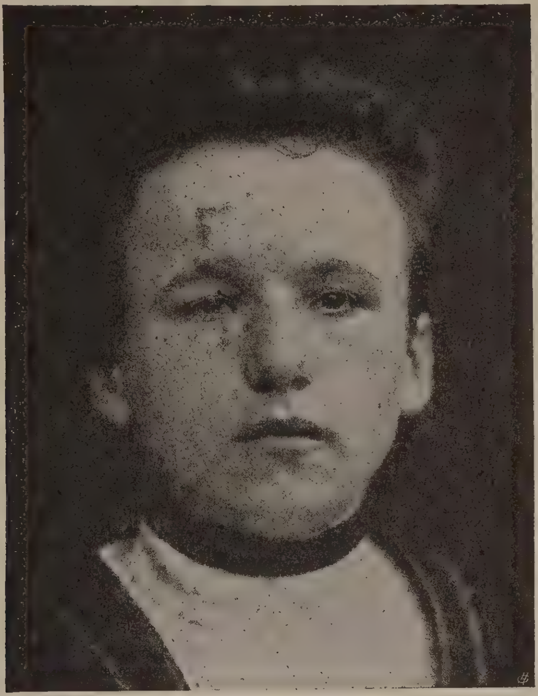


FIG. 116a.—Primary Position.

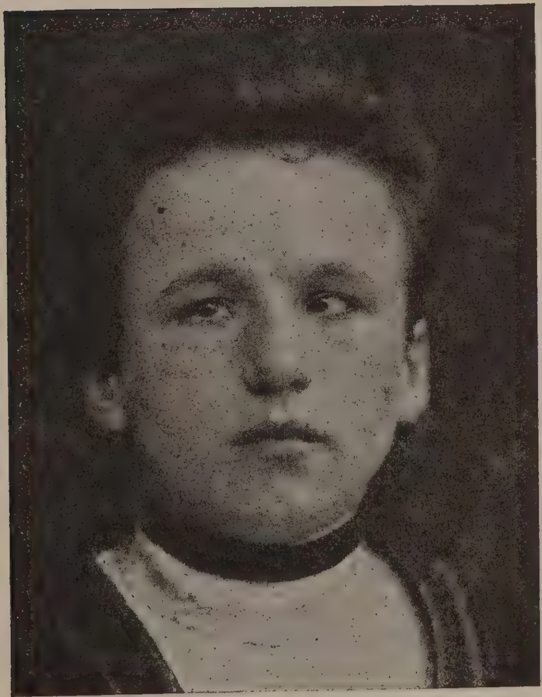


FIG. 116b.—Lagging Behind of the Right Eye. Paresis of the Right External Rectus

This is the first principal difference between paretic and muscular strabismus; the deviation is apparent in all positions of the eyes in the latter, while in the former it increases as the patient looks in a direction in which action of the paretic muscle is called for, but disappears whenever he looks in a direction in which this muscle is not used. If our patient looks to the left no strabismus can be perceived, because in doing this he does not bring his paretic externus into action.

Another important difference between paretic and concomitant strabismus is this: If we cover the healthy left eye of our patient and have him look at the light with his right, the covered left eye deviates inward much more than the right one did during fixation of the left. When he is compelled to look to the right with his weak externus he is obliged to send a more powerful impulse to this muscle than was necessary when the tract was intact. This stronger impulse is transmitted at the same time to the left internus, which is in a healthy condition, so the left eye is forced to turn inward much more strongly and the secondary deviation must be the greater.

The primary deviation is due to a lagging behind of the paretic eye, plus the tonicity of the antagonist of the paretic muscle, the secondary to a powerful muscular contraction, hence, when we find the secondary deviation to be greater than the primary we know the case to be one of paretic strabismus, of muscular paresis.

Now we come to a symptom that the patient himself noticed, the one that led him to consult us—*diplopia.* It is self evident that the pareses of the individual ocular muscles have their characteristic double images, and that the muscle affected can be determined from their position and displacement. There is no sense in trying to learn by heart all the possible positions of the double images —they will soon be forgotten—they are all governed by the same law that we learned in the study of concomitant strabismus:

A pathological position of convergence of the visual lines causes homonymous diplopia, a pathological position of divergence, crossed diplopia.

To demonstrate the position of the double images we place on our patient a trial frame, having a red glass before one eye to color the image of the light received by it. We will place the red glass before the right eye of our patient, and then he sees two lights—the red one to the right of the white one, which shows that he has homonymous diplopia. The explanation of this is that our retinal images originate from the opposite side of the room. If an object is situated to the right of the line of fixation of the right eye its image is formed on the inner portion of the retina. Inversely a stimulation of this inner side of the retina is localized to the outer side. In the position of strabismus a retinal image is misplaced and is localized by the part of the retina affected as if the eye were in its normal position. The right eye of this patient deviates inward, and therefore the image it sees must be projected to the right. When the eyes are divergent the double image is projected to the nasal side and the diplopia is crossed. We can therefore say that in paresis of an ocular muscle the double image is projected in a direction opposite to that in which the eye deviates, or, what amounts to the same thing, in the direction in which the affected muscle would normally act. We can analyze the double images in a case of paresis of an ocular muscle by means of this law.

The three final symptoms with which we meet in paresis of the ocular muscles are connected with the diplopia. This patient has stated that the images draw farther apart, or more closely together, according to the direction in which he is looking. External objects make apparent movements in front of his eyes, which sometimes make him quite uncertain in regard to them. A marked *feeling of giddiness,* which is very annoying, may be caused by this phenomenon.

The double images also result in *disturbances of orientation.* As the distance between them changes according to the direction of fixation, disappear in certain positions to reappear a moment later, the patient is finally unable to distinguish the real image from the false. One can easily fancy how troublesome this faulty orientation may be when such a patient has to descend a flight of stairs. This disturbance may be proved by the touch test suggested by *Graefe.* I cover the left eye of the patient, hold my finger in front of him, somewhat in the sphere of action of the paretic muscle, and ask him to grasp it quickly. He grasps to the right side of the finger.

We may imagine the cause of this faulty localization to be that when we wish to fix on an object situated considerably to one side we must innervate the proper muscles very strongly. Inversely we know that when we need to use a strong impulse of innervation in order to find an object, that object is situated far to the side. The idea that our eyes give us of its position depends in great part on this feeling of the impulse of innervation that is sent to them. Our patient is obliged to think that his paretic right eye has turned far to the right because he has used a strong impulse of innervation. He cannot know that the conduction is interrupted, that the eye is no longer under control. He believes that the fovea of his right eye, upon which the picture of my finger is cast, has been moved far to the nasal side by the outward rotation called for, and therefore projects the image of the object much too far to the right. We can say that the object fixed by a paretic eye is seen too far to the side toward which the paretic muscle normally turns the eye.

The final symptom of paresis of the ocular muscles is the *position of the head.* Our patient shows a certain predilection to keep his head turned to the right. He evidently tries to keep his head in such a position that the double images will give him the least annoyance, and this is attained when the paretic muscle is called upon to act little or not at all in the fixation of an object. If he turns his head to the right he can look straight ahead and to the left without calling the paretic externus into action. This position of the head is often so characteristic that the physician can make the diagnosis as soon as the patient enters the room. These form the cardinal symptoms of an uncomplicated case of paresis of the ocular muscles.

146. The Clinical Analysis of Paresis of the Extrinsic Ocular Muscles by the Aid of the Double Images, and the Most Important Diagnostic Signs

Our patient has complained for a long time of diplopia. Monocular diplopia is excluded, for it disappears as soon as one eye is covered, so we suspect the presence of a paresis of one of the ocular muscles, the cardinal symptoms of which are, 1, the impairment of mobility; 2, the paretic strabismus; 3, the diplopia; 4, the disturbance of orientation; 5, the feeling of dizziness; 6, the abnormal position of the head. When we look for these symptoms in this patient we find only the diplopia, for the impairment of mobility and the paretic strabismus are as good as absent. This teaches us that the paresis may be so insignificant that we cannot perceive it so to say macroscopically. In such a case as this the double images form the finest kind of a reagent by means of which to analyze the paresis.

I. Analysis of Paresis of an Ocular Muscle by Means of the Double Images

I can at least substantially demonstrate how we commence and proceed in such an analysis as this. We place on the patient a trial frame with a red glass in front of, for example, the left eye. He must hold his head still and follow the movements of the light by the movements of his eyes alone. He sees only a single light that has a rather reddish tinge everywhere, except when he looks toward the left; then he sees distinctly two lights, one red and one white, the red one to the left. Now we know that he has homonymous diplopia which appears when he looks to the left. We have learned that homonymous diplopia indicates a pathological position of convergence, hence this pathological position must be present when his visual lines are turned to the left. Two muscles take part in this movement, the internal rectus of the right eye and the external rectus of the left, so one of these two muscles must be paretic. The position of the double images furnishes the differentiation between them, for if the internal rectus of the right eye were paretic the eyes would be divergent and the diplopia would be crossed; hence it must be the external rectus of the left eye that is affected. If this is so the strabismic deviation must become greater the farther the left eye is turned in the sphere of action of its externus, even though it cannot be distinctly seen objectively. The farther this patient looks toward the left the greater becomes the distance between the double images and the more does the left eye lag behind in comparison with the right.

In the majority of cases of paresis of the ocular muscles we can test still better the behavior of the double images and the angle of strabismus if we do not move the object of fixation, but turn the head of the patient, according to the method of *Bielschowsky*. A light is placed in front of the patient at the distance of one metre and on a level with his eyes, which are kept constantly fixed upon it. If I turn his head to the left the muscles that turn his eyes to the right are called into action; if I bow his head the elevators act, and so on. When I turn his head to the left the distance between the two images decreases until they blend, but if I turn his head to the right the distance between them constantly increases, thus showing that one of the muscles that turn his eyes to the left is paretic. As the diplopia is homonymous the adducting muscle is stronger than the abducting one, and therefore it must be the left externus that is affected.

It is desirable to record the position of the double images in the different positions of the eyes. To do this we place the patient in front of the blackboard and mark on it the positions of the double images. It is not always as easy to demonstrate the symptoms of paresis of one abducens as it is in this patient. There are very mild cases in which the disturbance of equilibrium is kept latent by the involuntary effort of fusion and becomes manifest only in the peripheral portions of the field, i.e., when great demands are made upon the paretic muscle. The position of the head that is characteristic of paresis is absent in such cases. The primary deviation may be so slight, even in a total paresis of an externus, that it can be demonstrated only with the aid of the double images. We must therefore consider what is the cause of such variable behavior on the part of the primary deviation.

If a patient with paresis of the left abducens has homonymous diplopia when his eyes are directed straight forward, it follows that the visual lines intersect in front of the light, and that a pathological position of convergence is present, which can only be due to an overbalance of the left internus. This overbalance has frequently been spoken of as a secondary contracture, but this designation is only partially correct. We do not need much knowledge of the physiology of the action of muscles in order to be able to say that a contracture of its antagonist cannot take place the moment a paresis of an ocular muscle begins. We can speak of a secondary contracture only when the paresis is old, when sufficient time has elapsed to allow anatomical changes in the structure of the antagonist to have been formed. In its early stages the primary deviation, the preponderance of the antagonist, is caused by various nervous influences. The tonicity which, under normal conditions, maintains the tension of the ocular muscles is particularly to be taken into account. This tonicity may vary a great deal; if it is very marked the primary deviation is considerable, if it is slight the angle of strabismus is small.

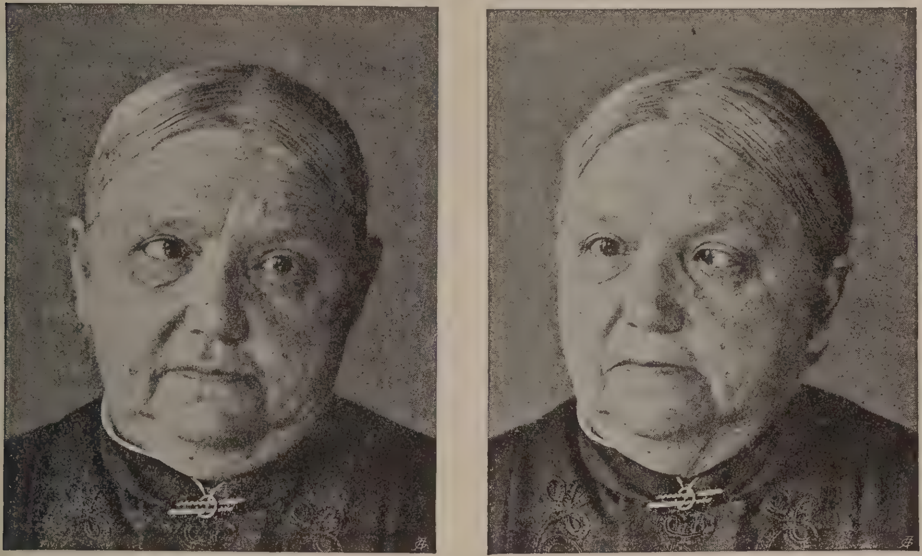


Fig. 117a. Fig. 117b.

Old Paresis of the Externus with Secondary Contracture of the Internus.

It may be mentioned as a diagnostic sign of the purely nervous influences which cause the primary deviation that *Bielschowsky* demonstrated an increase of the tendon reflexes in cases that had a great degree of strabismus. Other factors also have to be taken into account, such as changes in the tension of all the ocular muscles when one of them is stimulated. As I have said, we should speak of "secondary contracture" only in very old cases of paresis in which the field of excursion has been considerably reduced.

I wish to call attention to still another phenomenon that appears in the behavior of the double images as the direction of fixation is changed. When our patient looks farther and farther to the left his left eye lags more and more behind, and the distance between the double images continually increases. We have assumed such to be the constant behavior of the double images in order to facilitate comprehension, but the conditions are rather different in reality. At first the lateral separation of the images increases the farther he looks to the left, then the increase stops, the interval remains constant and finally, if the point of fixation is moved still more to the left, it decreases. The explanation of this is that the paretic muscle is still impelled by an impulse of innervation after the other eye has been prevented mechanically from responding to the innervation by the inhibitive effect of the ligaments of fascia, so that the paretic eye alone moves, and this must result in a diminution of the angle of strabismus.

Another point is this: Although the externus is purely a lateral rotator we often see a slight displacement of the double images. This is because the tendon of the externus is not always inserted with mathematical exactitude in the horizontal meridian of the eye, but a little above or below it instead, so as to exert a slight influence upon the vertical position of the eye and that of the double images.

The raising or lowering of the visual plane exerts little influence on a paresis of the lateral muscles; at the most the convergence decreases somewhat as the visual plane is raised and increases as it is lowered. Hence it is quite exceptional in a case of paresis of the abducens for a markedly lowered position of the head to be assumed in order to do away with the strabismic deviation.

A final point is that diplopia may be altogether absent in paresis of the external rectus. This is the case when the involuntary effort of fusion is able to keep latent the paretic convergence in a trifling affection, even though the vision of one eye has become very poor. Most persons with congenital paresis of the abducens have binocular single vision, because there is no contracture of the antagonist. They can maintain it throughout the greatest part of the field of fixation by a little turn of the head.

II. Diagnostic Signs of Pareses of the Extrinsic Muscles of the Eye

Paresis of the Rectus Superior

When the right superior rectus is paretic the visual line of the right eye is directed somewhat downward. If the patient looks up, the visual line of the right eye lags behind. This is most noticeable when he looks up and to the right, because the action of this elevator is greatest when the eye is abducted. When the right eye is compelled to fix by covering the left, the deviation upward of the visual line of the left eye is still more marked (secondary deviation). The double images are not of the same height, that belonging to the affected eye being the higher and at the same time converging toward the left. The difference in the height of the two images is greatest when the patient looks up and to the right.

Paresis of the Rectus Internus

A patient with a paresis of the right internal rectus has a tendency to keep his head turned to the left, because the activity of the muscle is thus avoided as much as possible and he is able to have binocular fixation toward the right. When he holds his head erect and looks toward the left his right eye lags behind and a strabismus divergens paralyticus results. When the left eye is covered and the right is compelled to fix, it makes a movement of adjustment toward the left, while at the same time the intact left eye assumes a greater degree of divergence, because the secondary deviation is greater than the primary. If the paresis is sufficiently marked, the backwardness of the right eye on looking to the left can be distinctly perceived. As this is a paretic divergent strabismus the images must be crossed, while the distance between them must increase toward the left and decrease toward the right.

Paresis of the Obliquus Inferior

When the inferior oblique of the right eye is paretic that eye must turn downward. It scarcely rises at all when the patient looks to the left and upward, while it exhibits hardly any backwardness when he looks to the right and upward. Of the two elevators of the right eye only the one is affected which is most powerful in action during abduction. The higher of the two images belongs to the right eye and is inclined to the right.

Paresis of the Rectus Inferior

When the inferior rectus of the left eye is paretic the visual line of that eye is the higher and its height must increase the more the eyes are turned down and to the left. The depression of the abducted visual line is prevented. The lower of the two images belongs to the left eye and is inclined to the right.

Paresis of the Obliquus Superior

When there is reason to suspect that one of the two depressors of the right eye is paretic we must remember that the eye in the position of adduction is depressed almost altogether by the superior oblique, while in abduction the depression is produced by the inferior rectus. If the right superior oblique is paretic the visual line of the right eye deviates upward when the head is held upright. When the right eye fixes, the left line of vision deviates downward to a greater degree (secondary deviation). If we have the patient look down, the right eye distinctly lags behind the other, most markedly when he looks to the left and downward, because in this position the right superior oblique, which depresses the eye when adducted, fails to act. On the other hand no fault in position is perceptible when he looks upward and to the left, or downward and to the right. The lower of the two images belongs to the eye that is deviated upward. The distance between the two decreases as the eyes are raised, increases as they are lowered, and is greatest when they are directed downward and to the left. In addition to this the patient states that the images are somewhat inclined toward each other, which shows that the vertical meridian of one eye has been rotated as the result of the deficiency of a muscle which acts, in part, to rotate the meridian.

All these various characteristics of the pareses of the individual muscles of the eye cannot and need not be remembered. It is only necessary to be familiar with the principle that underlies the examination in order to be able to deal with these cases. Every case of paresis of an ocular muscle can be accurately analyzed, although the conditions are by no means always as simple as in the examples that have been given. In the first place, the paresis may be combined, several muscles being affected at the same time, and they are often of so fleeting a nature that the conditions may change within a short time. After a paresis has been in existence long, atypical clinical pictures may be produced, which depend on the following phenomena.

The duties of fixation may be performed by the paretic eye, while the unaffected one may assume the secondary deviation of strabismus. Secondary contractures may form in the antagonists to the paretic muscles, while the paresis may increase or retrograde.

I have still to demonstrate a case that presents a condition which every physician is able to recognize—*complete oculomotor paresis.*

The upper lid hangs down loosely—ptosis due to paresis of the levator. We lift the lid so as to be able to see the eyeball, and then find that the pupil is dilated and does not react—paresis of the sphincter. The eye is adjusted for distance and cannot accommodate—paresis of the ciliary muscle. At the same time the eyeball protrudes somewhat from the orbit, because of the relaxation of the tonicity of the recti, which help to hold the eyeball in place. Finally the eye is deviated outward and a little downward because the two muscles that are not paretic, the external rectus and the superior oblique, draw the eyeball in this direction.

147. The Nerve Tracts for the Movements of the Eyes, and the First Clinical Problem in the Localization of the Focus of Disease in a Paresis of the Ocular Muscles

In a neurological sense, the diagnostic value of every analysis of a paresis of the ocular muscles is that in many cases the focus of the disease may be located accurately by its aid. We will try to solve this problem of localization in the case we have with us today.

The anamnesis furnishes nothing of importance. The patient is a strong man from the country, who smoked a good deal at one time until he had an attack of palpitation of the heart, with a feeling as of flies in his hands and pains in his temples. Improvement followed a reduction in the amount of his smoking. He is married and has healthy children. He shows no signs of syphilis and *Wassermann's* test is negative. Eight weeks ago he suddenly noticed that everything appeared indistinct when he tried to read with his right eye. An ophthalmologist found a paresis of the sphincter of his right eye with a partial paresis of the accommodation. He then had an attack of influenza that kept him in bed for several days and after his recovery he found that his visual trouble had grown worse. Things appeared still more blurred to his right eye and one day he noticed that he had diplopia. This brought him to us. Six weeks ago we found that he had perfect vision of both eyes with no changes in the fundus, paresis of the sphincter pupillæ and of the accommodation, together with ptosis and paresis of the superior rectus. The condition today is: Fundus, vision and visual field of both eyes normal; pupillary reactions of the left eye intact; almost total paralysis of the sphincter pupillæ and of the accommodation of the right. Of the extrinsic muscles of the right eye the following are paretic: 1, the levator, causing ptosis; 2, the rectus superior; 3, the rectus internus; 4, the obliquus inferior. A distinct advance in the paresis has taken place recently, for whereas at first only the muscles within the right eye were affected, now the levator, the superior rectus, the internal rectus and the inferior oblique are involved. I will now dismiss the patient and report the neurological condition.

A medium sized, strongly built man, with good muscles and strong bones, complains of diplopia. Tongue extended directly forward does not tremble and is freely movable. Hearing intact. All reflexes present, even the abdominal which are perhaps slightly weaker on the right side than on the left. Gross strength faultless. No ataxia. Tendon and skin reflexes all present. No disturbance of sensation. Pulse 72–80, strong, regular. Nothing in particular to be noted in regard to the internal organs. No trace of *Romberg*, hops backward with great accuracy, faces about with precision. Very much depressed at the time of the first consultation, rather querulous, tears in his eyes, worried about the future, very desirous of treatment. Today, after a good prognosis in the eye clinic, happy and cheerful, but always a critical judge concerning his own serious sickness.

If we are to try to locate the seat of the paresis we must review briefly two subjects: 1, We must know the course of the nerve tracts that have to deal with the movements of the eyes; 2, we must ascertain to which group of pareses of the ocular muscles this case belongs, for each group has a certain characteristic imprint according to the seat of the lesion.

I. The Course of the Nerve Tracts for the Movements of the Eyes

The movements of the eyes are under the guidance of various centres. The centres of higher rank are situated in the cerebral cortex and in the intracerebral tracts that join the cortex with the nuclei of the ocular muscles, and pass through the internal capsule and the tegmentum. It has been ascertained that associated movements of the eyes can be excited from areas of the cortex of the frontal, temporal and occipital lobes of both hemispheres.

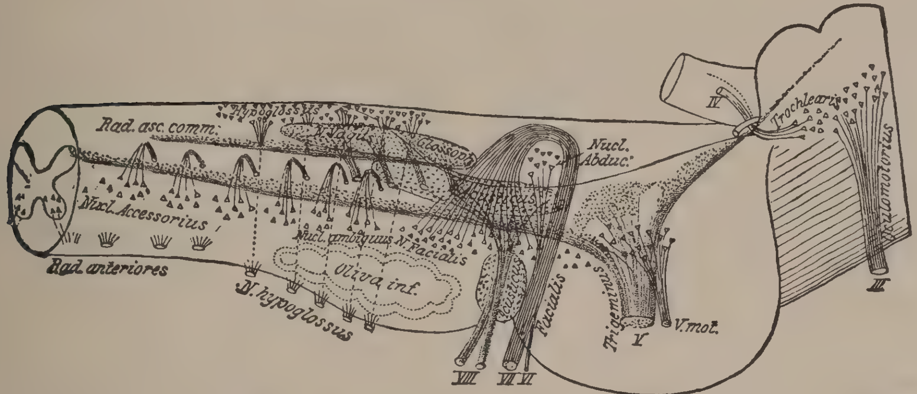


FIG. 118.—Nuclei of the Muscles of the Eye, after *Edinger*. (From *v. Mering's* "Textbook of Internal Medicine.")

The so called intermediate cells in the central tubular gray matter have an importance in the connection of the cortical tracts with the nuclei of the ocular muscles. This is the probable situation of the centres for the associated movements of the eyes. We know that the same tract runs through the posterior longitudinal bundle which regulates the concert of action of an externus with the internus of the other eye.

The centres of lower rank are the nuclei of the ocular muscles. They are situated beneath the aquæductus *Sylvii* and in the floor of the fourth ventricle on each side of the raphe. The one placed most anteriorly is the nucleus of the oculomotorius, which begins in the posterior part of the third ventricle and extends beneath the aquæductus *Sylvii* to beneath the posterior pair of the corpora quadrigemina.

Opinions are still much divided in regard to the details of the composition of this nuclear region. We do not yet know with certainty which of the individual groups of ganglion cells appertain to each of the muscles supplied by the oculomotor nerve. If we use as a basis the schematic delineation, as determined by *Bernheimer* from his researches on monkeys, we find that the nuclei of origin of those muscles that work in concert seem to lie together. The median nuclei appertain to the internal muscles of the eye. To these belong the paired, small celled median nuclei, which probably govern the movements of the pupils, and the single, large celled median nucleus, which is probably the seat of the innervation of the accommodation. On each side of the median nucleus are groups of ganglion cells which contain the origins of the nerves to the extrinsic muscles of the eye. A knowledge of the anatomical fact that the nucleus of the oculomotor nerve receives nutrition from two separate vascular regions is necessary in order to understand many of the pareses of the ocular muscles. The anterior portion receives its blood from the arteria cerebri posterior, the posterior from the arteria basilaris. The fibres that come from the nucleus of the oculomotor nerve pass downward through the crus cerebri, part going to one side, part to the other.

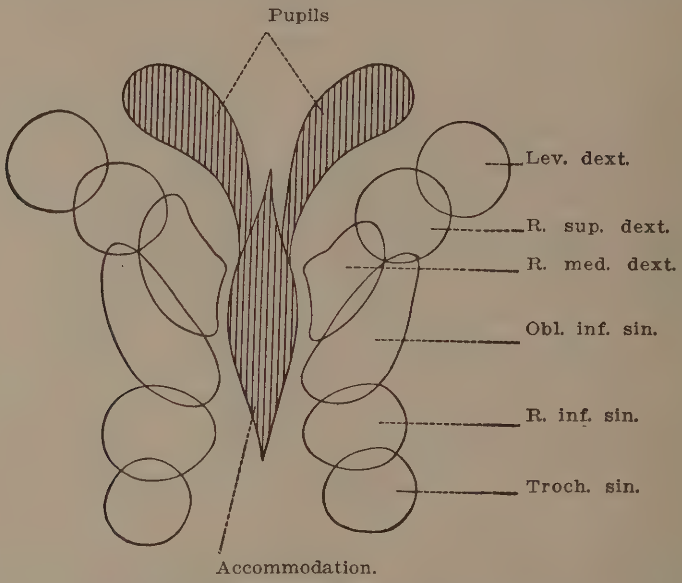


FIG. 119.—Accommodation. Schematic Delineation of the Nuclear Area of the Oculomotor and Trochlear Nerves, after *Bernheimer*.

The nucleus of the trochlearis is situated just at the posterior end of that of the oculomotorius, beneath the posterior corpora quadrigemina. The fibres of the trochlearis do not pass downward with those of the oculomotor nerve, but run in the opposite direction, upward and backward, into the velum medullare anticum, whence they pass to the other side, loop themselves about the cerebral peduncle and then pass to the base of the brain.

The nucleus of the abducens lies much farther back, a little in front of the striæ medullares, in immediate proximity to the nucleus of the facial nerve. The fibres of the abducens pass downward between the bundles of the funiculi pyramidales and reach the surface of the brain at the posterior margin of the pons. The trochlearis and abducens then pass forward and enter the orbit through the superior orbital fissure.

These anatomical conditions enable the nature of a paresis of the ocular muscle to be comprehended in many cases.

When all of the muscles of one or both eyes are paretic we call the condition ophthalmoplegia totalis. Ophthalmoplegia externa is more common; in this the extrinsic muscles are paretic while the sphincter pupillæ and the muscle of accommodation continue to perform their functions. The reason for this is that the nuclei for the sphincter and the ciliary muscle lie farther forward and may not be affected by the lesion. Inversely we meet with ophthalmoplegia interna when only the anterior segment of the nucleus of the oculomotor nerve has been injured.

II. To What Group of Pareses of the Ocular Muscles Does the Case Now Undergoing Examination Belong?

The site of the lesion may be anywhere along the entire nerve tract from the cerebral cortex to the peripheral termination in the orbit. In order to determine its situation in this particular case we need to inquire to which group it belongs. Pareses of the ocular muscles are divided into several groups for practical purposes, because they exhibit certain characteristics according to the site of the lesion, the most important of which are: 1, lesions of the centres of higher rank; 2, hemiplegia alternans; 3, orbital pareses; 4, basal pareses; 5, nuclear pareses.

Group 1. Lesions of the Centres of Higher Rank

It is very easily determined whether a lesion of the centres of higher rank is present, for experiment, clinical observation and pathology have taught us that lesions in these centres do not cause pareses of individual muscles, but of the associated or conjugate movements.

The Clinical Picture of Paresis of Associated Movements

The characteristic feature of this clinical picture is that the two eyes cannot be turned together in a certain direction. The concerted movement is absent, either up or down, to the right or the left, or the eyes may be unable to converge. Frequently the eyes move in the opposite direction. For example, if the eyes cannot be turned to the right when called upon to do so they may turn to the left, perhaps because of the preponderance of the other associated innervation, perhaps because of conditions of irritation. We call this condition conjugate deviation. In such a case the eyes are characteristically deviated toward the side on which the lesion is situated. For example, if there is a lesion in the right hemisphere which has destroyed the tract for the turning of the eyes to the left, the eyes are drawn to the right. If such a lesion causes a hemiparesis of the left side, not only the eyes, but the head is turned to the right, toward the site of the lesion. We must notice in the second place, in the examination of such a case, that the internus that is affected functionates very well in convergence, although it cannot act in the conjugate movement of abduction. This is because the tract for the innervation of convergence separates from that running to the corpora quadrigemina, which transmits the innervation for the lateral movements. This tract on the other hand may be affected alone, and then we have a paresis of convergence, while the lateral movement of the internus is intact. Such conjugate pareses, with or without conjugate deviation, occur with lesions in the crura cerebelli ad pontem, in the pons, in the corpora quadrigemina, and in the thalamus opticus. Exceptions may occur to this rule, that a lesion above the nuclei of the ocular muscles causes only conjugate pareses, for cortical lesions may cause an isolated ptosis, e.g., in depressed fractures of the vault of the cranium.

Group 2. Hemiplegia Alternans

The clinical picture of this paresis is characterized by the fact that an inability to look toward one side is associated with a paresis of the facialis or abducens on the same side, and of the limbs on the other. In such cases we suppose the lesion to be in the pons, because at this place the decussating tracts for the lateral movements of the eyes and the facial nerve lie close to the pyramidal tracts that do not decussate, and all these tracts may readily be destroyed by a single lesion.

Another form of hemiplegia alternans is that in which the oculomotor nerve of one side is affected at the same time as the limbs of the opposite side. In such a case we suppose the lesion to be in the lower part of the pedunculus cerebri. At this place the roots of the oculomotor nerve run close to the pyramidal tracts, which pass over to the other side lower down, so that a lesion in the crus of the cerebral peduncle will paralyze the oculomotor nerve of one side and the limbs of the other. It is easily determined in any particular case whether any such clinical picture is present. Our patient exhibits nothing of the sort.

Group 3. Orbital Pareses

Disease of the orbit must be present when the lesion is situated therein. We look for pain, which may be spontaneous, or may be induced by pressure either on the eyeball or on the margin of the orbit, for periostitis, or for inflammations starting from the accessory sinuses which may involve the nerves. Tumors must also be taken into account. But when there is no protrusion of the eyeball, no sensitiveness of the eye to pressure, and no choked disc, the pathological lesion that has caused the paresis is not in the orbit. The only question that can be raised is whether it may not be due to a peripheral neuritis, such as sometimes appears in the course of infectious diseases.

Group 4. Basal Pareses

We must determine whether the lesion is not situated at the base of the brain, where a great many morbid processes are met with, such as fractures of the base of the skull, the various forms of meningitis, tumors, periosteal troubles of manifold etiology, and vascular diseases. The following points in the differential diagnosis of pareses of basal origin may be mentioned. The abducens is particularly apt to be affected in fractures, as it is very easily injured at the apex of the petrous portion of the temporal bone. Affections at the base of the brain must be thought of first of all when pareses of other nerves appear in rapid succession following those of the ocular muscles, especially of the optic, facial, trigeminal, and olfactory nerves. Many cases are met with in which it is difficult to decide whether the paresis is basal or not.

When this question of differential diagnosis has been settled it still remains to be determined whether the condition is due to a lesion in the nuclei and roots of the ocular nerves.

Group 5. Nuclear Pareses

All that is necessary now is to bear in mind the anatomy of the nuclei of the ocular muscles that has already been described.

Characteristic Features of Nuclear Pareses

1. The presence of ophthalmoplegia interna indicates that the seat of the lesion is in the nuclei. If we find the pupil and accommodation paretic and can exclude a peripheral cause, such as the action of atropin, a nuclear affection is probable. If the ophthalmoplegia interna is of one eye alone, cerebrospinal syphilis is suggested immediately. The paresis may be for a long time the only symptom of this disease. When both eyes are thus affected we question whether the cause may not be ptomaine poisoning from meat. If the accommodation alone of both eyes is paretic, postdiphtheritic paresis of the accommodation is primarily suggested. 2. Bilateral ophthalmoplegia externa, paresis of all of the extrinsic muscles with the internal muscles intact, also indicates that the lesion is situated in the nucleus. 3. The same situation is indicated when isolated pareses of the ocular muscles occur in patients with a high degree of arteriosclerosis. The lesions in such cases are usually little hemorrhages by which the ganglion cells have been demolished. 4. Finally the lesion is situated in the nuclear region when, as in this patient, first the internal muscles and then the external muscles become involved systematically one after another in an order corresponding to the results of anatomical and experimental research.

If, on the other hand, all the branches of the oculomotor nerve on one side are paretic the site of the lesion may be elsewhere. The oculomotor nerve arises from cells of the same and of the opposite side. The lesion may be in the nuclear region in ophthalmoplegia totalis, or it may be at the basis cranii. Basal affections are met with even in pareses of single branches of the oculomotorius. The cause of an ophthalmoplegia totalis may also be found within the fissura orbitalis superior.

When all of these differential characteristics are taken into account no doubt remains that the lesion is situated in the nuclear region in this case. The diagnosis made in the neurological clinic was that of an encephalitis in the anterior portion of the oculomotor nucleus.

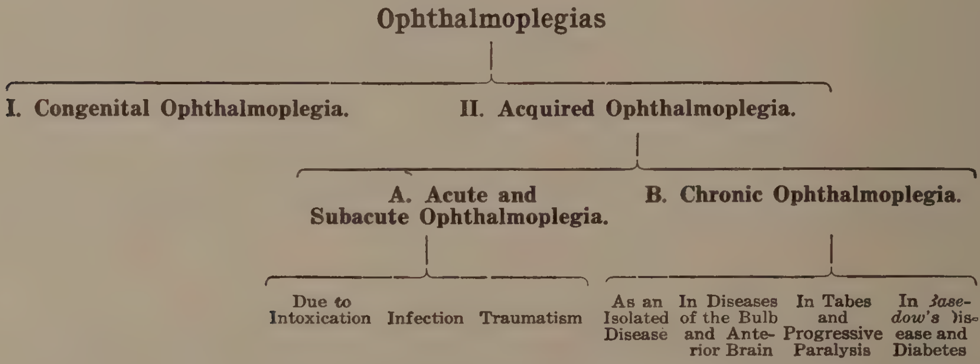
148. Etiology and Differential Diagnosis of the Ophthalmoplegias

We will now study another case of paresis of the ocular muscles, and bring out another point. We wish to know in every case of this nature not only which muscles are paretic and whether the paresis is of cortical, orbital, basal, or nuclear origin, but we wish also to try to ascertain something concerning its etiology from the peculiarities presented, whenever possible. The standpoint varies as the paresis is considered by the neurologist, or the ophthalmologist. In the eye clinic we first try to trace the etiology from the ocular symptoms themselves. For this purpose we gather together the factors of differential diagnosis that enter into the question. It is self evident that such a differential diagnosis, based wholly on the eye symptoms, has narrow limits, and that attention must be paid to the organism as a whole. We must learn to recognize these limits and therefore we will discuss the differential diagnosis of a paresis of the ocular muscles and see how far we can attain toward a diagnosis of its etiology.

This patient has a paresis of all the external muscles supplied by the right oculomotor nerve. The internal muscles of the iris and ciliary body are intact. The rectus externus and the obliquus superior perform their functions. I will omit the anamnesis and the description of the neurological condition, in order that we may have practice in the consideration of the most varying etiological factors.

This is a case of ophthalmoplegia, by which we understand a syndrome dependent on a disease of the posterior and middle brain, that is of the medulla oblongata, pons, floor of the aquæductus *Sylvii*, and posterior part of the third ventricle. Ophthalmoplegias may be due to the greatest variety of causes, and are of equal importance in ophthalmology and neurology. They may affect both eyes or only one. When all the muscles of the eye are involved we call it ophthalmoplegia totalis; when only the external muscles are affected, ophthalmoplegia externa; when the internal muscles alone are paretic we have either a bilateral or a monolateral ophthalmoplegia interna.

Although cortical and orbital pareses are comparatively easy to recognize the differentiation between a basal and a nuclear origin is frequently very difficult. The indications that the seat of the lesion is in the nuclear region have been given already, but when we are able to say that the lesion is at the nucleus our diagnostic duty is not completed, for we have still to trace the cause of the paresis. Therefore the question is raised again: What can the ophthalmologist contribute to the neurological diagnosis when we suppose an ophthalmoplegia to have a nuclear origin? I have drawn up the following table of the nuclear ophthalmoplegias to facilitate the investigation.


Ophthalmoplegias
I. Congenital Ophthalmoplegia.
II. Acquired Ophthalmoplegia.
A. Acute and Subacute Ophthalmoplegia.
B. Chronic Ophthalmoplegia.
Due to Intoxication
Infection
Traumatism
As an Isolated Disease
In Diseases of the Bulb and Anterior Brain
In Tabes and Progressive Paralysis
In *Basedow's* Disease and Diabetes

It is determined very quickly from the history whether an ophthalmoplegia of nuclear origin is congenital or acquired.

I. Congenital Nuclear Ophthalmoplegia

The congenital form presents the picture of ptosis and ophthalmoplegia externa with preservation of the innervation of the muscles within the eye. Heredity plays an important part in its causation, and it frequently happens that several members of a family exhibit analogous motor disturbances. It is apt to be accompanied by other congenital anomalies of the eye, such as nystagmus, or epicanthus, as well as by malformations in other parts of the body. Generally it is a sign that the paresis is congenital when the patient has no diplopia, and the secondary deviation of the other eye is imperfect when the paretic one fixes. Pathologically these congenital pareses are caused primarily by an aplasia of the nuclei of the ocular muscles involved. Congenital ophthalmoplegias differ from the acquired also in that they present a stationary clinical picture and are not the precursors or components of other diseases of the central nervous system, as is often the case with the acquired forms.

II. Acquired Nuclear Ophthalmoplegias

Our next question is: What can cause a nuclear ophthalmoplegia?

The large group of acquired ophthalmoplegias may be divided into two sections: the acute, or subacute, and the chronic, or chronically progressive, as a rule. Therefore we need at once to inquire: Does the ophthalmoplegia belong to the acute, subacute, or chronic form? The advantage we gain by this is that we are guided immediately to the etiology, for we must take into account such diseases as tabes, general paralysis, multiple sclerosis, diseases of the medulla, nuclei, and anterior brain, exophthalmic goitre and diabetes in a chronic ophthalmoplegia, while intoxication, infection, or traumatism is the cause of an acute or a subacute attack.

A. Acute and Subacute Ophthalmoplegias

The first question in a case of this nature is whether it is due to intoxication or infection.

1. Ophthalmoplegia from Intoxication

We have to take into account

a.—ALCOHOLISM

According to *Wernicke* the condition in these very serious cases is one of the consequences of a polioencephalitis acuta hæmorrhagica superior. The paresis is accompanied by grave cerebral disturbances, cloudy consciousness, delirium, dizziness, headache, atactic troubles and staggering. The disease is fatal in the overwhelming majority of cases. Numerous punctate hemorrhages may then be found in the region of the central tubular gray matter of the third and fourth ventricles and of the aquæductus *Sylvii.* The severity of the clinical picture, especially of the central general disturbances, is very marked. One characteristic of this form of ophthalmoplegia is that ptosis is comparatively often absent, and that the sphincter pupillæ and the accommodation remain intact, as a rule. We may conclude from this that the most anterior part of the oculomotor nucleus, in the posterior portion of the lateral wall of the third ventricle, is usually protected from the acute process. Sometimes we may find ophthalmoscopically optic neuritis with hemorrhages in the retina, but the paleness of the temporal side of the papilla should be particularly noticed; this is to be referred to the alcoholic amblyopia. According to *Uhthoff* this paleness of the temporal side of the papilla is to be seen in about ten per cent. of the cases of chronic alcoholism.

b.—OTHER FORMS OF INTOXICATION

Ophthalmoplegia is met with in rare cases of carbonic acid poisoning and after snake bites. It also occurs in chronic lead poisoning. The grave acute cerebral symptoms may be absent in these cases, while headache and convulsions may be present. The paresis differs from that of alcoholism in that the internal muscles may be involved. It is rarely that all the muscles of the eye are affected, and a neuritis can be found more frequently with the ophthalmoscope.

Botulismus is the principal one of these forms of intoxication that causes ophthalmoplegia. Grave conditions develop in the course of ptomaine poisonings by meat, sausages, fish, oysters, mussels, cheese, etc., in which pareses of the ocular muscles are prominent. The etiology of these forms of poisoning varies a great deal, but in true botulismus the condition is due to the virus of an anaërobic, spore bearing bacillus that was discovered by *van Ermengem* and has been named bacillus botulismus. I was the first in Germany to cultivate this bacillus from a case of ptomaine poisoning with ocular troubles caused by ham. This anaërobic bacillus in suitable nutritive media forms a dangerous toxine of enormous virulence.

The characteristics, on the part of the eyes, presented by this form of poisoning are as follows: Ophthalmoplegia interna; the pupils are dilated and do not react to light, the accommodation is paretic. This paresis of the internal muscles of the eyes is always bilateral, sometimes that of the accommodation remains partial. In addition these patients have ptosis of both upper lids, which gives them a peculiar appearance. Other muscles of the eye may or may not be involved, according to the severity and duration of the disease. The bilateral ophthalmoplegia interna with ptosis is the important feature. Morbid changes in the optic nerve and retina are absent. It is easy to recognize this form of ophthalmoplegia when the general symptoms of the intoxication are taken into account. The patients complain of depression, headache, and particularly of difficulty in swallowing and of dryness of the throat. They may have diarrhœa or constipation, and they have difficulty in breathing. The genesis of the disease has been explained by experimental research. *van Ermengem* was able to produce the dilatation of the pupils in monkeys by overfeeding them with the toxine of the bacillus botulinus, and then, together with *Marinesco*, found degenerations of the ganglion cells in various nuclei in the medulla. *Kempner* and *Pollack* likewise demonstrated signs of degeneration of the anterior horns of the spinal cord. I have proved that the ophthalmoplegia actually has its seat in the region of the oculomotor nucleus by experiments conducted together with *Stein*, in which we treated monkeys with toxoid holding botulismus toxines. We found distinct, discrete degenerations of the ganglion cells, especially in the single large celled median nucleus of the oculomotor nuclear region.

2. Acute and Subacute Ophthalmoplegias in Infectious Diseases

In the great majority of cases diphtheria causes simply a bilateral paresis of the accommodation. This is so common and so important a condition that we will devote special attention to it as soon as a case presents itself. We cannot pronounce this an ophthalmoplegia in the sense in which we are using the term. In the differential diagnosis of a bilateral, multiple paresis of the ocular muscles it is important to know that a marked ophthalmoplegia externa does occur after diphtheria, though rarely. One case of postdiphtheritic ophthalmoplegia may perhaps be met with among several hundred cases of postdiphtheritic paresis of the accommodation. It is characteristic of this form of ophthalmoplegia that the reaction of the pupil to light is preserved. Pareses of the abducens after diphtheria are more frequent than ophthalmoplegia. We explain the postdiphtheritic affection of the ocular muscles as due to a primary lesion of the nucleus. We shall return to this subject at another time.

Influenza may cause an ophthalmoplegia which is very similar to that produced by diphtheria. The external muscles of the eye and the accommodation may be affected, while the reaction of the pupils is retained, or, in other cases, the sphincter pupillæ may be involved. A bilateral paresis of the accommodation may be present alone, or associated with paresis of the sphincter. There is no certain rule in this respect as regards influenza. Cases of ophthalmoplegia of one eye following influenza have been reported. The reason the clinical pictures have so many forms is that both polioencephalitic processes and peripheral polyneuritic changes may be the cause of the paresis in influenza, while in diphtheria we have to suppose that the lesions are nuclear.

Other infectious diseases in which such bilateral ophthalmoplegias have been observed are typhoid fever, varicella, septicæmia, and miliary tuberculosis.

It is of diagnostic interest to know that such grave ophthalmoplegias are extremely rare after colds. Pareses of individual muscles, such as the abducens, are more common. Above all it should be strongly emphasized that an acute or subacute ophthalmoplegia due to syphilis is extraordinarily rare. It is quite otherwise in chronic progressive ophthalmoplegia, especially when the pareses are associated with other diseases of the spinal cord.

3. Acute Ophthalmoplegia after Traumatism

Finally we have to remember that occasionally hemorrhages or softenings in the region of the nuclei of the ocular muscles may be caused by traumatism to the skull, even without fracture or lesions at the base of the brain or in the orbit, and that these may produce an acute or subacute ophthalmoplegia. These nuclear pareses of the ocular muscles after injuries to the skull are certainly much more rare than those of a basal or peripheral origin, and in making this diagnosis we must exclude the presence of fracture of the skull and of basal pareses of other nerves. Bilateral paresis of the abducens is frequently of basal origin. In many cases late apoplexies following an injury to the skull may cause a nuclear ophthalmoplegia by producing foci of softening in the region of the nuclei.

Thus it is usually possible to determine the etiology by differentiation in a case of acute or subacute ophthalmoplegia, yet I cannot deny that there is quite a number of such conditions in which no etiological data can be secured, in spite of all our efforts. Sometimes the ophthalmoplegia exists alone, in other cases other pareses are added. We may then suppose that diffuse encephalitic changes, or hemorrhagic softenings are present, but an accurate diagnosis cannot be made. Still I believe that this schematic classification of acute and subacute ophthalmoplegias will prove useful in tracing back their etiology.

B. Chronic Ophthalmoplegias

If we are able to exclude acute or subacute ophthalmoplegia by the history and examination, the probability increases that we have to deal with a case of the chronic form, and the following is worthy of note from the standpoint of the ophthalmologist.

1. Chronic, Progressive Ophthalmoplegia as an Isolated Clinical Picture

A chronic, progressive ophthalmoplegia may occur without any complication on the part of the nervous system. The case is then one of bilateral ophthalmoplegia externa, with a moderate degree of ptosis, which begins in young persons and increases slowly, with remissions in the course of years. The ptosis does not always appear at the same time in both eyes. The branch of the facial to the eye is frequently involved. No morbid changes are to be found with the ophthalmoscope. This isolated and uncomplicated form of chronic progressive ophthalmoplegia is rare, and we have to question in any particular case whether the condition is not associated with other diseases of the central nervous system.

2. Chronic Progressive Ophthalmoplegia in Connection with Diseases of the Medulla and Anterior Brain

The association is most striking when the chronic ophthalmoplegia is combined with the diseases of the medulla and affections of the anterior brain. The most common cases are those in which symptoms of bulbar disease are present. In these the nuclei of the ocular muscles may be affected first and the troubles in mastication and swallowing appear later; the symptoms of bulbar paralysis then point the way at once to the diagnosis. In other cases symptoms of disease of the anterior brain, pareses of the limbs and of the muscles of the trunk, are added to those of the bulbar paralysis.

3. Connection of Chronic Ophthalmoplegia with Tabes and Progressive Paralysis

Chronic ophthalmoplegia is much more frequently a warning sign to the physician that other diseases of the central nervous system are present. Indeed we can say that this is true in most cases. *When we meet with a case of chronic progressive ophthalmoplegia we must think first of tabes,* for it has been proved that this is the accompanying disease in more than three fourths of all cases. Next comes progressive paralysis accompanying tabes in about 14% and occurring alone in about 7%. This fact is of the greater diagnostic importance, as the ophthalmoplegia sometimes precedes the appearance of the tabetic and paralytic symptoms by several years. Two other symptoms that the ophthalmologist has abundant opportunity to establish are the progressive atrophy of the optic nerve and the disturbances in the reactions of the pupils. Progressive optic atrophy occurs in more than a third of the cases of tabetic ophthalmoplegia, so a combination of these two conditions renders the diagnosis of tabes very probable. If in addition the patient has a reflex immobility of the pupils, the diagnosis of tabes is as good as positive, for this pupillary disturbance is present in more than three fourths of the cases of tabetic ophthalmoplegia. For this reason the condition of the optic nerve and of the pupil are of great value to the neurologist. Tabes leaves the other diseases of the spinal cord far behind as regards the frequency with which it is found in connection with progressive ophthalmoplegia, which occurs but rarely and exceptionally in syringomyelia, paralysis agitans and the combined systemic disease of the posterior and lateral columns of the spinal cord. It also plays a very subordinate part in disseminated or multiple sclerosis; according to *Uhthoff* it is met with in only about 2% of the cases.

If none of the causes that have been mentioned apply to the particular case the physician must remember that exophthalmic goitre and diabetes may give rise to such a paresis in rare instances, although the occurrence of nuclear ophthalmoplegia must be looked upon as exceptional in both of these diseases. Individual muscles are more commonly affected. Pareses of the ocular muscles occur in about 5% of the cases of diabetes. In rare cases a nuclear ophthalmoplegia may appear periodically; sometimes the recurrences take place at regular intervals. Finally I wish to emphasize the fact that a complete, purely unilateral ophthalmoplegia cannot be of nuclear origin, for it may now be considered as certain that the fibres of the trochlearis undergo a total decussation, while those of the oculomotorius decussate partially.

149. Acquired Ptosis and Its Differential Diagnosis

A short time ago this patient noticed a drooping of his upper eyelids, and now he has a ptosis of each eye. Hitherto he has been in perfectly good health.

By ptosis is meant a drooping of the upper eyelid due to a paresis of the levator palpebræ superioris, a muscle which is supplied by the oculomotor nerve. It may be affected alone, or in combination with other muscles of the eye, but the ptosis is always the most striking symptom.

Such a drooping of the upper lid may not be caused by a paresis of the levator, but may be due to other causes. Thus, trachoma may make the lid abnormally heavy by the thickening of the conjunctiva and so produce a sort of ptosis. Such a condition must be excluded primarily in making the diagnosis of paresis of the levator. We must also exclude a ptosis caused by an affection of another nerve. I have already spoken of *Horner's* symptom complex, in which a moderate degree of ptosis is produced by paresis of the oculopupillary fibres of the sympathetic, but it must be remembered that a paresis of the levator may be either organic or functional and that, before we can proceed to investigate the organic cause of a ptosis, a condition must be excluded which gives rise to a functional paresis. This condition is hysteria.

A lax ptosis is not rarely met with in hysterical persons who may present other ocular symptoms of hysteria, such as amblyopia and contraction of the visual field. This form may develop very gradually, or may appear suddenly, and is quite amenable to suggestion. Another form of hysterical ptosis is caused not by paresis of the levator, but rather by a contraction of the palpebral portion of the orbicularis. This form is known as ptosis pseudoparalytica, or spastica. Like the other it may be accompanied by other ocular symptoms of hysteria. A diagnostic point is that this spastic ptosis is almost always confined to one eye, while the relaxed hysterical form is usually bilateral. There is no reason to suspect the presence of such a functional, nervous disturbance in our present case, so we can now begin to inquire into the causes of the ptosis along the following lines.

Paresis of the levator may be of cortical, subcortical, nuclear, fascicular, peripheral, or muscular origin. It might therefore be questioned whether a sign could not be found in the clinical picture that would give an indication as to the site of the lesion, from which we might be able to determine the etiology, but this anatomical way is only partially useful to the practical diagnostician, as I can show by an example. It is to be assumed a priori that the levator palpebræ has a cortical centre, for its function is intimately associated with the activity of the cortex of the brain. Every morning, on awakening, the eyes open, the light enters and then the activity of the cerebral cortex begins. *Horsley* and *Schaefer* have proved by experiments on monkeys that a centre for the movements of the eyes is situated in the first and second frontal convolutions of these animals, but we know nothing accurately as yet in regard to the cortical centre in man, for even the numerous cases in which circumscribed lesions of the cerebral cortex have been verified by autopsy have given us no positive information as to its locality. Probably it lies in the superior parietal convolution, or angular gyrus. It is extremely uncertain that a purely cortical ptosis has ever been seen, and our knowledge concerning the subcortical tracts of the levator is still more inaccurate. We may therefore exclude this group from our differential diagnosis. We cannot determine from an acquired ptosis alone whether it is of muscular, fascicular, peripheral, or nuclear origin, we have to seek assistance from the accompanying conditions. It can only be said in general terms that a complete ptosis is usually the result of peripheral paresis, and that we frequently meet with nuclear pareses in incomplete ptosis. Distinguished neurologists maintain that we have as yet no absolutely positive indications of a nuclear paresis, and we must not forget that in most of these cases it is much more important to the patient that we should diagnose the nature of the disease, rather than its situation. Every candid physician knows that much that is proudly termed diagnosis in the field of the central nervous system is only conjecture. If, in spite of these difficulties, we wish to analyze the condition of ptosis accurately and utilize it for purposes of differential diagnosis, we must consider the case clinically as well as anatomically, and ask: What diseases are chiefly responsible for the development of ptosis? I have combined these two principles in the plan of differential diagnosis that I have outlined and will follow in the examination of our patient.

Differential Diagnosis of Ptosis

1. Organic paresis of the levator or not? 2. Ptosis, congenital or acquired? 3. Ptosis due to acute intoxication or infection? 4. Due to injury? 5. Due to syphilis? 6. Due to some other disease of the central nervous system? These are the six major questions to be discussed in the differential diagnosis.

First question: Organic paresis of the levator or not? We have to decide whether we can exclude a functional disturbance due to hysteria.

If the case is one of true ptosis due to organic disease we come to the second question: Is the condition congenital or acquired? We shall soon study congenital ptosis and learn its operative treatment. It is practical common sense to ask the patient this question at the outset, for the congenital form is the most common type of paresis of the levator. The differentiation is thus made very quickly.

Third question: Has the patient been suffering from an acute intoxication, or infectious disease? Whenever I am called upon to investigate a case in which the levator is paretic I ask this question as soon as the fact of paresis has been established, because it always seems natural to do away at once with the factors that are rare and in all probability not to be taken into account.

Ptosis in Infectious Diseases

This is met with very rarely. When only a single case due to diphtheria occurs among over 1,000 cases of that disease, and among more than 150 cases of postdiphtheritic paresis of the accommodation, the diagnostic value of the symptom is not great. Postdiphtheritic paresis in the eye usually takes the form of paresis of the accommodation with the sphincter not involved. The external rectus is the most frequently affected of the extrinsic muscles of the eye.

The condition is similar in epidemic cerebrospinal meningitis, the abducens is frequently affected, the levator rarely. Ptosis is likewise extremely rare after influenza, measles, typhoid fever, pneumonia, and rheumatism. A sort of ptosis can be produced in facial erysipelas by the increase in volume of the lid caused by extension of the inflammation to the skin, but a paresis of the levator is rarely seen. The preceding infectious disease gives us the clue in all of these cases.

Ptosis in Intoxications

Ptosis occurs in quite a number of cases of polioencephalitis hæmorrhagica superior. An acute ophthalmoplegia externa develops in chronic alcoholics, together with stupefaction and delirium, to which may be added optic neuritis, or partial optic atrophy. In such a case the history and the general appearance indicates the cause at once. Another form of acute poisoning in which ptosis frequently occurs in connection with ophthalmoplegia interna is botulismus, or ptomaine poisoning, which we have studied already. It is met with much more rarely in lead poisoning.

Ptosis after Traumatism

Fourth question: Has the patient suffered an injury? When, from the statements of the patient, an injury appears to have been the cause of the ptosis we follow the anatomical path of investigation, for a traumatic ptosis can be caused only by a direct injury to the levator, or to the orbital branches of its nerve. I cannot specify all kinds of injuries, but will simply mention a few examples.

When a contusion, like the blow of a fist, falls upon the upper lid, the muscle and its nerve may be crushed and so a ptosis may be caused. The muscle itself, or its insertion into the tarsus, may be divided in stab or shot wounds. Hemorrhages beneath the skin, or entering the upper lid from the orbit, may press upon the levator or its nerve. Such hemorrhages in the orbit may originate from a laceration of the orbital vessels, or from a fissure of the orbital wall. In the case of an isolated fracture of the wall of the orbit, or of a fracture of the base of the skull, the blood oozes through the orbital fat forward and appears first as an ecchymosis in the conjunctiva. In such a case the blood may exert so much pressure on the muscles and nerves as to produce a paresis of all the external muscles of the eye, while the internal ones remain unharmed. The suffusion of the conjunctiva and of the lids appears sometimes several days after the injury, and sometimes quickly.

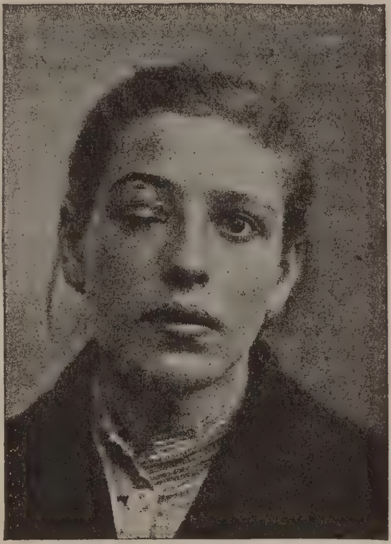


FIG. 120.—Ptosis after Abscess of the Lid.

Paresis of the levator, usually associated with that of the superior rectus, caused by delivery with forceps is orbital in most cases, but it may be due to traumatic lesions of the trunk of the oculomotor nerve, or of its cortical or nuclear centres.

The oculomotorius may be injured in the cavernous sinus when the internal carotid is torn in a fracture of the base of the skull; a pulsating exophthalmos with ophthalmoplegia and ptosis then develops. The oculomotor nerve itself may be torn at the base of the skull, as it is considerably endangered in its course along the petrous portion of the temporal bone, so that it, as well as the abducens, may be crushed in a fracture of the base through this region. Even if it is not torn the trunk of the oculomotor nerve may be compressed by hemorrhages in such a fracture, and it happens not so very rarely that a ptosis with dilatation of the pupils, paresis of the hypoglossus and paresis of the pharyngeal reflexes, calls attention to the fact that a fatal purulent meningitis has developed.

Occasionally a ptosis and other pareses of the ocular muscles may have a nuclear origin after an injury to the skull. Foci of softening are frequently formed after such injuries. Punctate hemorrhages which turn into softenings may be situated in the region of the nuclei of the nerves of the muscles of the eye, but, to diagnosticate a traumatic nuclear paresis as the cause of a ptosis, an orbital paresis and a fracture of the base of the skull must be excluded. It is an interesting fact that diabetes forms an additional symptom of such a traumatic nuclear paresis.

It is a matter of great legal importance that late traumatic apoplexies can take place a long time after an apparent recovery from a grave injury to the skull.

Finally, the cortical centre of the levator can be affected in a traumatic softening of the cortex of the brain. Such cases have been observed in which ptosis has followed a depressed fracture of the vault of the skull. It is worth taking the trouble to determine whether the development of a ptosis is due to an injury or not.

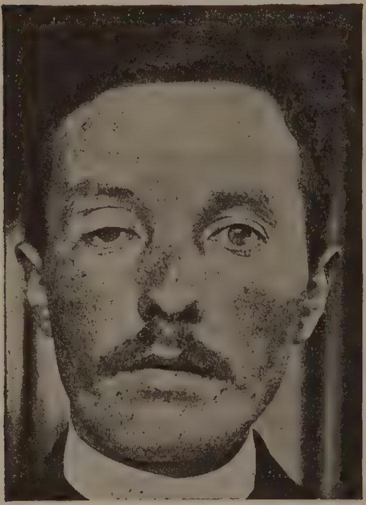


FIG. 121.—Ptosis of One Eye.

Fifth question: Is syphilis the cause of the ptosis? I advise that this question be considered as soon as acute infection, intoxication and injury have been excluded. This is the disease by which the oculomotor nerve is most frequently affected. If any indication of syphilis is present we see rise before our mind's eye the entire tract of the oculomotorius, for the lesion may be situated at any point from one end of the tract to the other, and there are many symptoms to which the ptosis may call our attention. Clinical and pathological research has given us the following:

Ptosis Due to Syphilis

The swelling and weight caused by a tarsitis luetica may cause the lids to droop; this is a sort of pseudoptosis. Gummatous periostitis of the margin of the orbit is much more common. As such a swelling develops it causes the lid to droop, partly by the thickening produced, partly by pressure upon it. If the gummatous mass extends more deeply into the orbit, involves the muscles and compresses the nerves, an exophthalmos with restricted mobility is formed; hence we must think primarily of syphilis when we see such an exophthalmos with ptosis. If the gummatous process extends into the depths of the funnel of the orbit, where the motor nerve trunks lie close together, all the nerves of the eye will be affected. It is therefore important to know that an isolated total paralysis of the oculomotor nerve, to which our attention is called by the ptosis, cannot be due to an affection in the orbit if the trochlearis and abducens are unimpaired.

Another favorite seat of syphilitic inflammation is the tissue in the superior orbital fissure, where the nerves that pass through the oculomotorius, the trochlearis, the abducens, and the upper branch of the trigeminus, may receive exceedingly severe injuries. In such cases a complete ptosis suddenly appears, together with paresis of the internal and external muscles of the eye and a loss of the sensitiveness of the cornea, conjunctiva, and other regions supplied by the first branch of the trigeminus. As the process takes place near the optic foramen it is apt to involve the optic nerve and to cause amblyopia or amaurosis. Exactly the same clinical picture may be produced when a gummatous basal meningitis extends to this place. The physician should always think of periostitis in the superior orbital fissure whenever the ophthalmic branch of the trigeminus is involved, together with the oculomotorius, trochlearis, abducens and opticus, while the infraorbital nerve is unharmed. The ptosis is always complete and is never absent when the oculomotor nerve is involved in any of the diseases of the orbital fissure.

The most common form of cerebral syphilis is the gummatous meningitis of the base of the brain. We must therefore consider whether the ptosis and its accompanying syndrome is not caused by this disease. The trunks of oculomotor nerves coming from the brain have been found to be affected by an independent gummatous neuritis and perineuritis, but basal meningitis plays a part in this. I strongly advise this disease to be borne in mind when signs of paresis of the cranial nerves, especially of the oculomotor and optic, appear among general cerebral symptoms, weakness of memory and disturbances of consciousness. A gummatous basal meningitis is generally located in the deep recess between the chiasm, the peduncles and the trunks of the oculomotor nerves. In fresh cases the optic and oculomotor nerves are enveloped in a gelatinous mass which becomes more indurated later on. It is therefore easy to understand that the trunks of the oculomotor nerves on both sides are usually involved, and that a bilateral ptosis calls for a differential diagnosis in this direction.

Finally, ptosis may be caused in cerebral syphilis by a lesion in the region of the root and nucleus of the oculomotor nerve. In the region of the root the trouble is usually due to gummata or softenings in the peduncle. The nuclei of the ocular muscles are predisposed to syphilitic disease because of its blood supply. It has been proved that a disease of the basilar artery may very easily cause nutritive disturbances in the region of the nucleus. Gummata may also grow in this region. Even a primary atrophy of the nuclei of the ocular muscles has been observed by *Oppenheim* during the virulent stage of syphilis.

It is by no means easy to diagnosticate a nuclear paresis in a given case. The advance of our knowledge has greatly disconcerted the theories formerly held. *Mauthner* said that the progressive paresis of the muscles of the eye had a nuclear cause in most cases. We know now that a gummatous lesion in the trunk of the oculomotor nerve alone can cause an isolated ptosis. This does away with the old dogma that a disease of a nerve trunk must cause a paresis of all the muscles supplied by it. We also know that a ptosis which remains isolated need not be of fascicular, nuclear, or cortical origin, for it has been satisfactorily proved that such a permanently isolated ptosis may appear in diseases of all places along the course of the oculomotor nerve.

The conditions in cerebral syphilis are quite different from those in tabes. When we meet with an isolated ptosis in the latter disease we can ascribe it confidently to a degeneration in the nucleus of the levator, while in cerebral syphilis it may be caused by a basal affection. It is only when the isolated ptosis occurs together with a hemiparesis of the opposite side that the lesion is situated either in the pons or in the peduncle. When the ptosis is not isolated, but is associated with pareses of other muscles, the disease may also be situated at the base of the brain. So many cases with all possible combinations of ophthalmoplegias have been observed in basal affections that this situation of the syphilitic disease should be thought of first. The lesion can be nuclear only when the internal muscles of the eye remain intact from the beginning, when the muscles supplied by the oculomotor nerve gradually become paretic one after another, when the pareses are bilateral, when sugar appears in the urine, and when the disease has remissions. A nuclear paresis cannot be present when a paresis of one or both sides of the body coexists. In such cases the cerebral peduncle is involved. A nuclear paresis is also contraindicated when the optic nerve is affected and a neuritis, or an atrophy can be detected with the ophthalmoscope. Temporal hemianopsia indicates that the chiasm is involved, while homonymous hemianopsia is a sign of a lesion in one tract. Outward from the nuclei the supranuclear and cortical regions of the oculomotor nerve come into consideration, but a supranuclear ptosis is not met with in this disease, for cerebral syphilis attacks by preference the base of the brain and causes peripheral nerve affections through the meningitis. Supranuclear pareses of the ocular muscles are usually conjugate paralyses. The cortical region of the levator still needs investigation, as I said at first. Whatever may be the difficulties in the way of an accurate diagnosis one thing is certain: In no other disease is bilateral paresis of the oculomotor nerve so common as in syphilis, and it is almost the rule that the levator of one or both eyes is paretic in grave cases of cerebral syphilis with involvement of the oculomotor nerve; a sufficient reason why this disease should be taken into account in the analysis of an acquired ptosis.

Sixth question: Is any other disease of the central nervous system present which may be the cause of the ptosis and of the symptoms that may accompany it? In addition to the ones that have been mentioned there are numerous other diseases of the central nervous system to which a ptosis may direct our attention. This is another reason why we should try to trace the differential diagnosis. After these three classes of cases have been excluded we know that the cause must be one of these other diseases. We will first consider the rare affections.

Isolated Bilateral Ptosis

Isolated bilateral ptosis is a rare condition, which was first described by *Fuchs*. A bilateral paresis of the levator appears in persons who are in perfect health, almost exclusively in old women, remains isolated and gradually increases in degree. The movements of the eyeball and the reactions of the pupil are normal. The ptosis becomes greater when the patient is tired, and a marked contracture of the frontal portion of the occipitofrontalis on each side calls attention to the condition. The way in which the lax skin is drawn in beneath the margin of the orbit is striking. It is not yet known whether this is due to a nuclear affection, or to a primary atrophy of the muscles.

Ptosis in Chronic Progressive Ophthalmoplegia Externa That Remains Isolated

We also meet with a curious form of ophthalmoplegia associated with ptosis, a bilateral progressive paresis of the extrinsic muscles of the eye. It begins in early childhood, advances slowly with no other disturbances in the central nervous system and without fever, pauses at times in its development, but usually results in total ophthalmoplegia externa. The pupils, accommodation, vision, and fundus remain normal. The disease frequently begins with a ptosis of one or both eyes and may last thirty or forty years under certain circumstances. This form of ophthalmoplegia is primarily differentiated from the congenital impairments of mobility by the fact that congenital pareses remain absolutely stationary. The etiology of this disease also is unknown.

In syringomyelia the abducens is the most apt to be affected of all the ocular muscles because its nucleus is most deeply seated, but a ptosis caused by paresis of the levator is rarely met with. A ptosis due to paresis of the sympathetic is on the contrary common, for paresis of the sympathetic on one side, with lesions of the oculopupillary tracts is a well known symptom of this disease.

Ptosis in Tabes

We come now to a disease in which ptosis is of greater diagnostic importance, tabes. It has long been known that reflex immobility of the pupil is one of the most important symptoms of this disease, although the close relations of the reflex immobility to the oculomotor nerve are as yet insufficiently understood. Next to the reflex immobility of the pupil come pareses of the extrinsic muscles of the eye; they are present in about 20% of the cases and frequently form the first signs that are to some degree distinctive of the disease. The levator is the muscle that is particularly apt to be affected, so we have to think of tabes when we meet with a ptosis and must always be on the lookout for this indication. An isolated ptosis of one or both eyes often appears in the initial stage, but it does not remain isolated, as a rule. It either retrogresses, for the pareses of tabes have the general character of being transitory, or it becomes associated with reflex immobility of the pupil, or with some other symptom, such as absence of the patellar reflex, optic atrophy, or lancinating pains. This is the condition in which we find our patient, in whom a reflex immobility of the pupil can be demonstrated. An isolated complete paresis of one oculomotor nerve is rarely met with in tabes, an ophthalmoplegia externa is rather more frequent, as it occurs in from three to five per cent. of the cases. The transient appearance of the ptosis is usually surprising. These pareses are apt to disappear quickly, even without treatment. In this respect they differ from the affections of the optic nerve, which are of a progressive character. Cerebral syphilis must be carefully excluded when the diagnosis of tabes is based upon ptosis, either isolated or associated with ophthalmoplegia externa. The differential diagnosis between cerebral syphilis and commencing tabes is apt to be very difficult and is sometimes impossible, for syphilis, especially of the base, may cause an isolated ptosis, and is very often the cause of extensive pareses of the muscles supplied by the oculomotor nerve. One point is of interest. In commencing tabes reflex immobility of the pupil is usually present, in cerebral syphilis we frequently find absolute immobility of one pupil with paresis of the accommodation. Still reflex immobility of the pupil has been observed in cerebral syphilis. We also occasionally meet with ptosis together with paresis of the abducens in the prodromal stage of tabes. The site of the tabetic pareses is in the nuclei of the ocular muscles, the high degree of degeneration of which has been shown by *Keimerling* and *Baedeker*.

Ptosis in Progressive Paralysis

Pareses of the ocular muscles are much less common in progressive paralysis; they occur in only two or three per cent. of the cases that are not complicated with tabes. Therefore ptosis is rare and is not of great diagnostic importance.

Ptosis in Multiple Sclerosis

Ptosis occurs in about 10% of the cases of multiple sclerosis, and may be of importance in the diagnosis, which is frequently very difficult at the beginning of the disease. We also meet with ptosis in the chronic and subchronic ophthalmoplegias found in the diseases of the medulla oblongata and anterior brain. Ptosis of both eyes associated with weakness of the muscles of mastication and of the throat is a typical symptom of asthenic bulbar paralysis. It is likewise a symptom of great importance in cases of hemorrhage into the brain, in which *Willbrand* and *Saenger* find that it has been observed in 75%. It is caused by hemorrhages at the base, in the cerebral peduncles, in the tegmentum, in the thalamus, and in the nuclear region of the oculomotorius. It appears, almost as a rule, whenever embolic and thrombotic softenings of the brain are associated with pareses of the muscles of the eyes. When it is found in connection with a hemiplegia of the opposite side it indicates that the region of the cerebral peduncle is the site of the lesion.

The condition of ptosis is also of considerable importance when the possibility of an abscess in the brain is being considered. It is particularly apt to be present when the abscess is located in the temporal lobe. I have diagnosed correctly such a location of an abscess of the brain from a ptosis and mydriasis to which were added in succession on the opposite side a paresis of the facial supply to the mouth, then a paresis of the arm and finally of the leg. It is never a symptom of abscess of the cerebellum, which is so often caused by diseases of the ear.

The conditions in meningitis are such that ptosis is very frequent only in the tubercular form, in which disease it develops very gradually, the eyes at first are opened more slowly and then by degrees it becomes more distinct. Oculomotor pareses are very rare in epidemic meningitis, the abducens is more commonly affected. Purulent meningitis affects the convexity of the brain, as a rule, so it is only when it happens to be situated at the base, as the result of an infectious disease, or of an inflammation of the ear, that ptosis can occur. The syndrome of sinus thrombosis is frequently completed by a ptosis.

When tumors are situated at the base of the brain they frequently give rise to early focal symptoms because of the ease with which they may compress the cranial nerves. When they are situated in the sella turcica they cause trouble with the muscles of the eye and with the trigeminus, as well as produce hemianopsia, so it is of importance in the differential diagnosis that this combination of symptoms is not produced when the situation of the tumor is intracerebral, though ptosis and other symptoms on the part of the ocular nerves may be excited by its distant action. The accurate localization of a tumor of the brain is possible only in rare cases. Tumors of the hypophysis are betrayed by the bitemporal hemianopsia, to which may be added amaurosis of one or both eyes, caused by optic atrophy. Paresis of the oculomotor nerve on the same side as the tumor and paresis of the facial and hypoglossus on the opposite side is characteristic of tumors of the peduncle. When the neoplasm is situated in the region of the nucleus of the oculomotor nerve we usually have a bilateral ptosis. In tumors of the pons we have paresis of the facial on one side and of the extremities on the other, but paresis of the abducens of one side with paresis of the extremities of the other may be present. Ptosis has been observed in connection with tumors of the cerebellum, when they exerted pressure upon the corpora quadrigemina. I mention all this simply because a tumor of the brain should be borne in mind when we are studying a case of ptosis.

This completes our excursion into differential diagnosis, which was started by our case of acquired ptosis, and we have now to decide what the cause is in this patient. The differentiation does not happen to be difficult because of the presence of the other characteristic symptoms of tabes. Large as is the field that the diagnostician has to hasten through, I hope that I may have facilitated at least the first steps in the differentiation of the causes of a paresis of the levator by the questions: 1, Intoxication or acute infection? 2, Injury? 3, Syphilis? 4, Some other cerebrospinal disease? As a single symptom ptosis is decisive as to the etiology of the disease in a particular case in only the rarest of instances, but as we meet with it in such an enormous number of diseases it seems seasonable to me that we should have had this practice. Practice makes the master in differential diagnosis.

150. Congenital Ptosis and Its Operative Treatment

A glance at this patient reveals that he has a ptosis of the upper lid of one eye. The history indicates that the condition is congenital, and I present the case for two reasons.

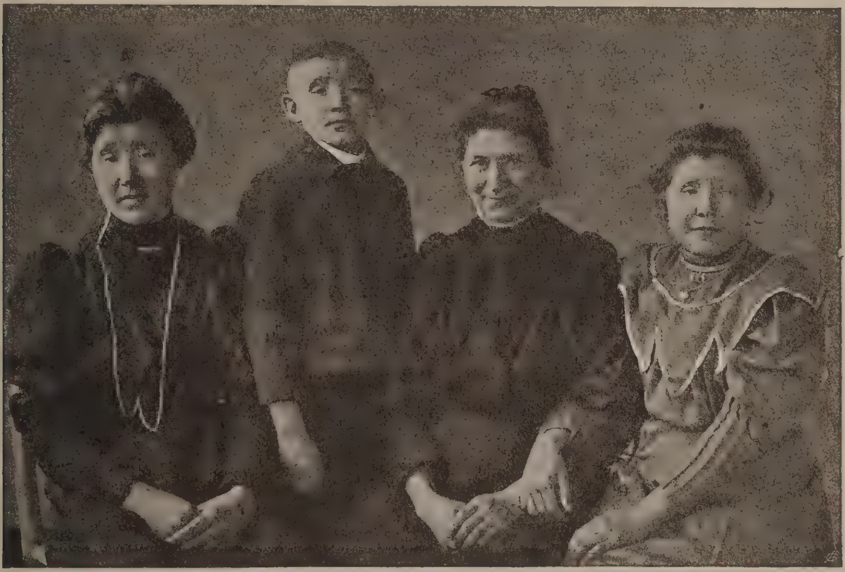


FIG. 122.—Family with Congenital Ptosis.

In the first place, congenital ptosis exhibits a number of clinical characteristics which are so peculiar that we need to be acquainted with and to recognize a certain clinical picture.

In the second place, congenital ptosis is the form of paresis of the levator in which we are most frequently able to intervene with operative measures, while in other forms an operation must be looked upon as a last resort, in accordance with the well recognized law concerning pareses of the ocular muscles.

I. The Clinical Picture of Congenital Ptosis

Congenital ptosis is usually bilateral and incomplete, giving the patients a peculiar, sleepy expression. It is met with in every possible degree. If the paresis is only slight, so that the lids droop only a little, the vision is not interfered with. It may also affect one eye alone, as in our patient, in whom the upper lid hangs down loosely and completely covers the pupil.

When the patient tries to lift the lid he succeeds slightly by means of a contraction of the frontal portion of the occipitofrontalis. The skin of the lid is drawn somewhat upward by the lifting of the eyebrows, so as to open the eye a little. A distinct interspace can be seen between the lower margin of the eyebrow and the margin of the orbit. The covering fold, which can be distinctly perceived on the normal eye above the sulcus orbitopalpebralis, is more or less spread out. Parallel folds of skin, with the convexity upward, indicate where the frontal portion of the occipitofrontalis is contracted. The surface of the forehead is lessened by the formation of these folds in the skin. The eyebrows are displaced upward and somewhat outward by the action of the frontal portion of this muscle, so that their nasal ends are rather far apart. The longer the extent of the skin of the upper lid the less efficient is the action of this muscle. The patient must bend his head backward if light is to enter his eye through the slight enlargement of the palpebral fissure thus produced. The constant contraction of the frontal portion of the occipitofrontalis causes a peculiar expression of the face from which, in connection with the drooping of the lid, any case of ptosis may be readily recognized. It is an interesting fact that these contractions, which have a teleological sense, develop gradually as the intelligence of the child increases. During the first months of life such eyes are scarcely opened at all, and then the action of this muscle is utilized by degrees. The paralysis of the levator may be complete or incomplete, the same as that of other muscles of the eye, and the efficiency of the contraction of the frontal portion of the occipitofrontalis is dependent on the length or shortness of the skin of the lid. If the skin is lax and long this contraction may not avail sufficiently to correct the anomaly, even when the ptosis is of low degree, while on the other hand, when the skin is short and the action of the muscle strong a paresis of the levator may appear to be incomplete even when it is total. As the effect of an operation depends to a considerable degree on whether it is complete or not, we need to determine the degree of paresis when the action of the occipitofrontalis is excluded. The simplest way to do this is to press with the finger upon the upper margin of the orbit and fix the skin, so that the action of the occipitofrontalis is prevented. When we do this we find that our patient is able to raise the lid but a very little. Congenital pareses of the levator are not always as marked as this. In many the fault can scarcely be observed when the eyes are in their ordinary position and the patient has to look up in order to render the paresis distinct.

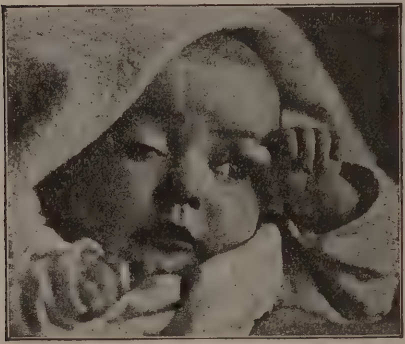


FIG. 123.—Ophthalmoplegia in Infancy. A Child Eight Months Old Helping Himself by Lifting the Lid.

Many patients with congenital ptosis have other motor defects of the eyes as well, most frequently upward. This is partly due to the fact that such patients are not accustomed to raise their eyes, but the superior rectus is sometimes less efficient than it should be because its insertion is placed too far back. Defective motility of the muscles supplied by the oculomotor nerve, or of all the muscles of the eye may be met with. The accommodation and the movements of the pupil are always normal. Strabismus is common. The vision is often poor, as the result of congenital amblyopia, errors of refraction and nystagmus. Malformations of other parts of the body and mental defects frequently accompany bilateral congenital ptosis. Epicanthus, by which is meant a fold of skin covering the inner canthus on each side of the bridge of the nose, is particularly common.

Congenital ptosis may occur in an isolated case, or it may be transmitted more or less through several generations. The hereditary form is usually associated with other motor defects of the extrinsic muscles. The causes of congenital ptosis are many. According to *Ammon* a ptosis will result if the upper lid is so hypertrophic that even a well developed levator is too weak to raise it. Adhesions between the muscles and abnormal insertions have been found, and the muscle is sometimes replaced by a band of connective tissue. In still other cases the levator may be completely absent, or the oculomotor nerve may be imperfectly developed, or wholly wanting. Such cases have been useful in revealing the construction and function of the nucleus of the oculomotor nerve. *Willbrand* and *Saenger* have demonstrated an aplasia in this nucleus in congenital ptosis.

A word in regard to the differential diagnosis. A case like this is easily recognized, as a rule, but we must remember that a slight ptosis may be simulated when the formation of the skull is asymmetric and the opening of one orbit is larger than that of the other, for the distance from the eyebrow to the margin of the lid must be greater when the circumference of the opening of the orbit is greater. It must also be differentiated from the peculiar condition presented by an isolated progressive ophthalmoplegia externa. The presence of congenital motor defects in the muscles of the eyeball serve to indicate its congenital nature. There are no secondary contractures, such as are apt to form in acquired pareses, and there is no diplopia. Moreover, in congenital pareses the muscles are likely to cause backward movements of the eyes, but isolated, chronic, progressive ophthalmoplegia is so rare that the differentiation is not difficult, as a rule.

II. Treatment of Congenital Ptosis

The treatment of ptosis is imperfect because we cannot furnish a perfect substitute for the physiological action of a muscle. A number of spectacle frames have been devised, by means of which the paretic lid may be held up, but frequently they are of little use. The cosmetic disfigurement remains, and for this reason we are obliged to operate in many cases.

A large number of operations have been devised from the most divergent points of view. An old method was to shorten the lid by an excision of a fold of skin, frequently with a portion of the orbicularis so as to weaken at the same time the antagonist to the paretic levator. Other operations have been based on the principle of strengthening the weak, but not totally incapacitated levator. The advancement of this muscle, as recommended by *Everbush*, is for this purpose. The skin is incised, the fibres of the orbicularis separated so as to lay bare the upper margin of the tarsus, several loops of thread are passed as high as possible through the tendon of the levator, their ends carried between the skin and tarsus, brought out in the intermarginal space and tied. The threads should be carried as closely as possible to the tarsus, in my opinion. The tendon of the levator is drawn down in this way, but unfortunately the result is only temporary in many cases.

Another method of strengthening the levator is to shorten the tendon and muscle. An incision is made at the convex margin of the tarsus, the skin is dissected up from the subjacent tissue as far as the eyebrow, the fascia tarsoorbitalis is divided, so as to expose the tendon of the levator, and a portion of the muscle is resected. The end of the shortened muscle is then sutured to the convex margin of the tarsus. The results produced by this operation are likewise only moderate.

These methods cannot accomplish the desired purpose if the ptosis is considerable; hence a number of operations have been devised to substitute the action of another muscle for that of the levator. Thus *Motais* sutures a portion of the superior rectus to the upper margin and anterior surface of the tarsus, while others try to substitute the action of the frontal portion of the occipitofrontalis.

The desired result may be obtained in suitable cases by sutures as recommended by *Pagenstecher* and others. One needle of a thread armed with needles at both ends is introduced beneath the free margin of the lid, passed beneath the skin to a point above the eyebrow, where it is brought out through the skin of the forehead. The second needle is passed parallel to the first at a short distance from it. The threads are then tied firmly over a pledget of gauze and are allowed to remain until they have given rise to the formation of firm cicatricial bands that unite the lid with the frontal portion of the occipitofrontalis. Good results may be obtained in this way in the milder degrees of ptosis, but two, three or more sutures must be introduced.

Another operation of this class is that of *Panas*, in which the union between the lid and the occipitofrontalis is secured by means of a pedicle taken from the skin of the lid, passed beneath the eyebrow and fastened to the skin of the forehead. A good elevation of the lid can be obtained in this way, but the cosmetic effect produced by the scars, which are considerable and run vertically to the course of the orbicularis, is not good.

The best operation for ptosis is the one devised by my teacher, *Hess*. After the skin has been thoroughly disinfected and the eyebrow shaved we make an incision exactly in and for the entire length of the eyebrow. The lower margin of the wound is seized with forceps and the entire skin is undermined down close to the margin of the upper lid. The bleeding may be fairly free, and it is essential for a good final result that this be arrested by pressure or a tampon. We thus have two wound surfaces opposed to each other and can displace the skin of the upper lid. Next we introduce three sutures armed with a needle at each end by passing the needles through the skin at about the middle of the lid and bringing them out about 2 cm above the eyebrow. When these threads are drawn upon, the skin is folded so that the covering fold of the normal eyelid is simulated. The threads are then tied over rolls of cotton, or glass beads. The excellent effect produced by this operation is due to the fact that the adhesion of the wound surfaces naturally holds the upper lid in position better than sutures alone, while in addition a union is effected with the frontal portion of the occipitofrontalis by the cicatricial bands that develop along the sutures. Another advantage of this operation is that we are able to regulate the effect during the first day or two by tightening or loosening the sutures. It is necessary to try to get the greatest possible primary effect, because this retrogrades somewhat later. Careful asepsis must be maintained in the after treatment, to guard against an infection in the comparatively large cavity of the wound. During this time the cornea must be kept well protected with ointment.

151. Torticollis and Paresis of the Trochlearis

The history of this boy, now 10 years old, is that he had a severe general, febrile disease about five years ago. According to the statements of his mother he has ever since been accustomed to carry his head in a peculiar, wry position, on account of which he has received many whippings. He was subjected to orthopædic treatment without avail. If we take hold of his head and place it upright it returns to the oblique position as soon as we let go. He inclines his head to the left shoulder, lowers his chin and rotates it at the same time to the left. As all the means tried to correct this torticollis have been in vain, and as the child complained one day of his eyes, his mother has brought him to us.

I will now demonstrate that this peculiar position of the head has hitherto been the only means by which this tormented child could do away to some extent with the disturbing effects of a grave paresis of the right trochlear nerve, which supplies the obliquus superior. This is learned from an analysis of his symptoms.

First I wish to mention again that in the consideration of the action of the superior oblique only that portion of the muscle is to be taken into account which extends backward and toward the temple from the trochlea at an angle of about 50°, to be inserted beneath the superior rectus, chiefly in the temporal portion of the eyeball. By its contraction it depresses and abducts the visual line and inclines the upper pole of the eye to the median side. The depressing action is the one of these three components which is of most interest to us just now. This is slight when the visual line is directed straight ahead, as it forms an angle of 50° with the plane of the muscle, and is less, or nothing, when the eye is abducted, when the only effect produced by the muscle is to roll the eye inward, but when the eye is very strongly adducted it is considerably depressed by the contraction, because the visual line coincides with the plane of the muscle. This boy has an inclination of the head by means of which he can fix a light placed in front of him with his visual lines elevated and turned to the right. As he is so partial to this position of his eyes we are justified in the suspicion that a depressor of the eye is paretic, one also that is most efficient when the affected eye is turned to the left. Such a depressor must be either the left inferior rectus, or the right superior oblique. How are we to determine which muscle is paretic, and whether one or more are affected?

We know that in paresis of the ocular muscles the secondary deviation of the unaffected eye is greater than the primary deviation of the other and that by this means we can perceive in which eye the paresis exists. Therefore we straighten up the head of the boy and direct him at the same time to look at a candle placed in front of him at a level with his eyes. When we cover the right eye the left remains quiet, but when we cover the left the right makes a distinct downward movement. Hence while the left eye was fixed the right deviated upward a little. But if the left eye is observed behind the covering hand, or better when the hand is removed, it is very distinctly seen to be deviated downward to a greater degree than the right was formerly deviated upward. The secondary deviation of the left eye is the greater, and therefore the paresis must be in the right. As we are justified in suspecting a paresis of a depressor we now test the movements of the two eyes in comparison with each other. It is seen at once that their position is normal in looking to the right and downward and that the right eye plainly lags behind in looking to the left and downward. Hence it follows that the depressor of the right eye which is paretic is the one in which the depressing component of its action is most marked when the eye is adducted. That muscle is the superior oblique. The other movements of the eyes present no abnormality, except that when the child looks horizontally to the left the vertical strabismus of the right becomes more distinct. This is readily understood, because when the superior oblique of the right eye is paretic the inferior oblique must preponderate in the position of adduction and incline the eyeball upward.

The behavior of the double images likewise proves the right obliquus to be paretic in this case. If we place a red glass before his right eye and ask him whether he sees two when he looks at the light with his head in its characteristic position, he says no, but as soon as we straighten up his head he sees double. The image pertaining to his right eye is the lower and is rather oblique, inclining to the right, which shows that the paretic muscle must act to roll the eye inward and therefore must be either the depressor of the right eye, the obliquus superior, or the levator of the left, the rectus superior, but we need only to note the position of the double images in the various parts of the field of fixation to obtain a definite differentiation. If we depress this boy's head, so as to elevate the line of fixation, the double images grow closer together until they blend, but when we raise his head and so depress the visual lines the lower image sinks more and more, and if we turn it to the right the obliquity of the image increases. The vertical separation of the double images is greatest in the left lower quadrant of the field of fixation. Taking this in connection with our finding that the mobility of the right eye is limited on looking inward and downward, no doubt is left that this boy has a paresis of his left trochlearis. So much as to the way in which to proceed in the analysis of such a case as this.

The relations between paresis of the trochlearis and habitual torticollis are of more interest. This boy inclines his head to the left shoulder, depresses and rotates his chin to the left, because it is only in this position that he can fix with both eyes and avoid the annoying diplopia. He does not see double when the line of sight is raised and turned to the right, but as soon as his head is held upright by means of an orthopædic device, or otherwise, he sees double and is deprived of the only means at his disposal to avoid the annoyance thus caused. So we cannot blame him that, in spite of whippings and other measures, he continually reassumes this position. He inclines his head to the left in order to bring the object he is looking at into a portion of the field of fixation where the paretic muscle is called upon to act little or not at all. In paresis of the abducens the patient turns his head toward the side of the affected muscle; in paresis of the trochlearis, on the contrary, the habitual lateral inclination of the head is toward the healthy eye, as in this case, for with his head turned to the left the rotators of the eyes to the left, of which the obliquus superior dextra is one, are disburdened, while the rotators to the right are innervated. In this comparatively simple way the boy is able to render parallel the meridians of the retina which are correspondingly inclined in consequence of the paresis.

This typical inclination of the head is not present in all cases of paresis of one trochlearis. It is absent when one eye is very amblyopic, and when the angle of strabismus is so great that binocular vision is impossible even when the head is held in this manner, but, according to *Bielschowsky*, the best authority concerning this condition, this habitual torticollis is present in 20% of the cases. Hence its presence should call attention to the possible existence of such a paresis. The diagnosis is facilitated when it is borne in mind that of the three components into which the functions of the superior oblique can be divided one cares particularly for the depression of the adducted eye, and that therefore the vertical component of the angle of strabismus is the most important. This vertical divergence is increased by the predominance of its antagonist, the inferior oblique and its secondary contracture. Next to the vertical component, the differentiation depends on the disturbance of the balance of the muscles that rotate the vertical axis of the eye. As soon as one of the upper muscles, which act to rotate the vertical axis of the eye inward, becomes paretic the tonicity of the lower muscles, which rotate it outward, deviates the upper pole of the eye to the temporal side. The inclination of the double images toward each other is therefore one of the most important symptoms of which we need to take notice. A number of other interesting symptoms are met with. It is not necessary that the oblique image should appertain to the paretic eye, it may belong to the other. When the paretic eye has the better power of vision it may assume the duty of fixation. All of these conditions must be investigated very carefully if we are to adopt correct therapeutic measures. We have to determine whether the advancement of the antagonist, or the tenotomy of the rectus that acts in the same way in the other eye, is indicated.

This case is one of habitual caput obstipum caused by a paresis of the trochlearis. The condition was named by *Cuignet*, in 1873, torticollis oculaire. It hardly seems necessary to emphasize the fact that it is not every habit of holding the head obliquely met with in children which is to be ascribed to such a paresis. A cramped position in writing may accustom children to an oblique position of the head, entirely aside from the surgical causes of caput obstipum. We also know that astigmatics whose cylindrical glasses are not accurately adjusted, can correct the erroneous position of the axes by a lateral inclination of the head, but I think that use may be made of this demonstration with advantage, for the knowledge that a paresis of the trochlearis may cause such a habitual wry neck is essential to enable the right treatment to be applied in such a case.

152. Paresis of the Accommodation after Diphtheria

The paresis from which this boy is suffering is one that every physician should know and recognize. He tells us that he has been unable to read or write for several days past because his near vision is so bad. His eyes are emmetropic, have normal vision for distance, and the ophthalmoscopic condition is perfectly normal, therefore his inability to see near objects clearly must be due to a fault in his accommodation. The reaction of the pupils to light is perfectly normal, the sphincter pupillæ and its innervation are intact, but when we test the range of accommodation we find it to be considerably less than it should be in a normal eye at his age. Hence he has a partial paralysis of the accommodation of equal degree in the two eyes.

When a child with such a partial paralysis of the accommodation comes under observation the first thing to be thought of is diphtheria. Closer inquiry into the history reveals that this boy had a sore throat that kept him in bed a few days about three weeks ago, but that his parents did not consider it anything serious. This typical case is of interest in more than one respect. From the results of our former study, in which we analyzed the differential diagnosis of ophthalmoplegias, there can be no doubt that we have to deal here with a case of ophthalmoplegia interna following diphtheria. What should the physician know of this paresis of the internal muscle of the eye, and what is to be done for the child?

Bilateral paresis of the accommodation is very common after diphtheria. Figures vary, but we may say that it is demonstrable in about 10% of the cases. It characteristically appears during convalescence on the average from three to four weeks after the beginning of the infection. The children are apt to find that they read and write with difficulty on their return to school. Another characteristic of the disease is that the paresis of the accommodation is of equal degree in the two eyes. A third characteristic is that the sphincter pupillæ remains intact in most cases. It is quite seldom that the pupils react slowly, or not at all, to light. A fourth characteristic is that the paresis does not appear suddenly, but develops gradually, requires several days to reach its maximum, and that even then the disability of the ciliary muscle is not total.

The severity of the trouble depends on the degree of paresis and not a little on the optic construction of the eye. Hypermetropic children, who form the majority, have to accommodate more strongly than others, and naturally are more affected than others by a loss of accommodative power, while a slight degree of paresis of the accommodation may not be noticed by myopic children.

It is a very remarkable fact that paresis of the accommodation may follow very mild attacks of diphtheria, so mild that sometimes they completely elude observation, and it occasionally happens that the appearance of the paresis is the first evidence presented that a child has been infected with diphtheria. In a comparatively large percentage of these cases we find a nasal speech due to a paresis of the soft palate. Pareses of other muscles of the eye and signs of ataxia are more rarely met with.

Infections with the true diphtheria bacillus alone are able to cause such a paresis of the accommodation; streptococcal anginas never produce it. Hence it is due to the virus of the diphtheria bacillus. The greatest variety of lesions have been found in these postdiphtheritic pareses, some in the peripheral nerves, some central, some in the vessels.

I believe that we must have recourse to the researches concerning the diphtheria toxin in order to understand this paresis of the accommodation. We know, from the classical studies of *Ehrlich*, that the diphtheria bacillus produces two different substances, the toxin, which is held responsible for the acute clinical symptoms, and the toxon, by means of which he was able to produce paresis experimentally. The latter acts much more slowly than the toxin, so I believe that the paresis of accommodation after diphtheria is due to the effect of a toxon, that the seat of the lesion is in the nucleus of the muscle of accommodation, and that it consists of a specific lesion of the ganglion cells which acts in a way similar to that in which paresis of accommodation in botulismus is produced. This theory has the support of an important clinical symptom. Experimental research has proved that the affinity of the specific antitoxin to the toxons is less than to the toxins, and that the paresis of the accommodation is not reduced in the least by treatment with the curative serum. We may therefore suppose that antitoxic serum can influence with great difficulty and insufficiently the action of the toxon in the human organism.

The practitioner should be acquainted with this disease, particularly that he may reassure the parents, who are usually greatly worried, for the prognosis is perfectly good; recovery takes place spontaneously in the course of a few weeks. No treatment is needed, except that when it is absolutely necessary that the children should study we may prescribe glasses to be worn for a while in order to make the work easy. I cannot confirm the statement that the paresis passes off more quickly when such glasses are worn. The ciliary muscle itself is healthy, and spontaneous recovery follows the restitution of the impaired innervation.

A word concerning differential diagnosis. Paresis of the accommodation may occur in other diseases, like syphilis and diabetes, but usually botulismus is the one that needs to be differentiated. Patients suffering from botulismus may complain of throat trouble, but as has been said their pupils are paretic and dilated at the same time.

153. Recurrent Oculomotor Paresis

This young woman has suffered since childhood, and has always been under medical treatment for attacks of migraine, characterized by headache confined to one side followed by vomiting, which have occurred every four or five months. Until two years ago the headaches and vomiting constituted all of her trouble, but since that time a paresis of the ocular muscles has accompanied each attack, and at present she has a total paresis of the right oculomotor nerve.

I am gratified to be able to exhibit this case for three reasons: First, it presents the picture of total paresis of the oculomotor nerve, as the condition is now at its height; second, this ophthalmoplegia is evidently connected with the migraine and is therefore not solely of ophthalmological interest; third, this symptom complex of recurrent oculomotor paresis is so rare and peculiar that it is worth while to ponder a little over it.

Before we can recognize such a rare clinical picture, before we can diagnose it as a *recurrent* oculomotor paresis, we must be able to exclude all of those diseases in the course of which repeated attacks of paresis of the oculomotor nerve may take place. The most important of these are tumors of the brain, cerebral syphilis, tabes, multiple sclerosis, hysteria, chronic meningitis, the chronic ophthalmoplegias that remain isolated, and aneurysms at the base of the brain. Sometimes the differential diagnosis is very difficult. In this case we have been able to exclude by a thorough examination all of these diseases, and the diagnosis of recurrent oculomotor paresis is justified by the facts that the attacks recur periodically, that they always affect the same side, and that they regularly begin with the symptoms of migraine followed by paresis of the oculomotor nerve alone.

This disease almost always affects only one and the same eye. It may be divided into three periods: that of the pain, that of the peculiar paresis, and that of the interval between the attacks. The symptoms begin with a dull, violent pain which spreads over the occiput and brow, and presents the characteristics of migraine. The pain is always on the same side of the head as that on which the oculomotor nerve subsequently becomes paretic. The duration of this period of migraine varies a great deal. Cases have been observed in which it lasted only one day, and others in which it continued for weeks. The length of the attack also varies in the individual recurrences. After the pain has abated the oculomotor paresis appears, it may be suddenly, or it may reach its acme by degrees. Sometimes it becomes total at once, sometimes it does not become total until after several attacks. In other cases again the paresis always remains incomplete. Its duration varies. Cases have been reported in which it was present for only a few days, and others in which it lasted for weeks and even months. Both mild and grave forms are met with. The levator is involved in the great majority of cases, so that the ptosis calls our attention to the condition. Another point of interest is that the involution of the paresis of the individual muscles supplied by this nerve differs, that of the internal muscles of the eye taking the longest time. After this period comes the interval, during which the patient may be perfectly well, with no trace of the paresis. In other patients the first attacks alone are so benign; the greater the number of attacks the more readily do the paretic conditions of the muscles supplied by the oculomotor nerve persist during the interval to be simply increased by the next. The length of the intervals likewise varies greatly; usually they last for several weeks, sometimes for several months.

Although the paresis of the ocular muscles is the most prominent symptom of this disease, and frequently is the only one present, yet other symptoms accompany it in many cases. Pareses of the abducens, trochlearis and facialis have been observed. The trigeminus may be involved, and then we find hyperæsthesia, especially of its first branch. Concentric contraction of the visual field has been noticed on the paretic side. Many patients complain also of tinnitus and deafness of the ear on the same side.

The cause and the true nature of this disease are wholly unknown. As it bears a certain relation to migraine, *Charcot* named it migraine ophthalmologique, and as the connection has been attested by other investigators we are obliged to consider it a special form of that disease. According to *Moebius* the pain is to be explained by a disease of the nuclear region. Granted an œdema of the nuclear portion of the oculomotor nerve, the irritation produced would extend to the root of the trigeminus and might give rise to headache and vomiting. The pain is not neuralgic, because it does not radiate along the course of a nerve. Still, there are features in this clinical picture which do not harmonize readily with the conception of migraine, for instance, the fact that the pareses may persist during the interval. It may also be pointed out that migraine occurs chiefly in the well to do class of people, that it rarely appears before puberty, and that it gradually grows milder to disappear entirely in old age. Recurrent oculomotor paresis on the contrary chiefly affects persons of the lower classes, usually begins very early and grows more severe with advancing age. The scintillating scotomata which so frequently accompany migraine are exceedingly rare in recurrent oculomotor paresis.

A great variety of views are held in regard to the seat of the lesion. According to *Moebius* the symptoms are indicative of a nuclear paresis, according to *Mauthner* and others they point rather to a basal disease. I think that more attention should be paid to the examination of the blood in these cases than has been in the past, for I can detect conditions which lead me to suspect that the cause of this peculiar, periodical affection may be found in a disease of the blood, but this is something that I cannot prove.

The final result in most cases is a more or less complete oculomotor paresis, but sometimes the attacks cease. In two cases, reported by *Richter* and *Karplus*, a tumor was found in the trunk of the oculomotor nerve. Whether the etiology of the disease is to be explained in this way must be left for the future to determine. We are confined therapeutically to symptomatic treatment.

154. The Form and Width of the Palpebral Fissure under Normal Conditions, and the Oculopupillary Symptoms of Paresis of the Sympathetic Nerve

The first thing noticed in the examination of this patient is that the right upper lid hangs down a little lower than the left and covers the cornea to the upper margin of the pupil, but the skin of the lid is not relaxed and is without folds; it is rather drawn in toward the orbit. The lid still retains its motility. Convex furrows are to be seen in the skin of the forehead over the right eye. The palpebral fissure is narrowed not only by a drooping of the upper lid, but also by an elevation of the lower, as can be seen if we look carefully. The affected side of the face is warmer and redder than the other. The right pupil is smaller than the left, but still reacts to light.

This peculiar syndrome, first described by *Horner*, is due to a paresis of the oculopupillary fibres of the sympathetic. The course of these fibres to the eye has been described already, so I will only repeat that those which supply the dilatator pupillæ, the orbitalis and the palpebralis superior and inferior, come from the sympathetic of the head and neck.

The most marked symptom presented by a comparison of the two eyes of our patient is the narrowness of the palpebral fissure. There is a slight degree of ptosis, but if the correct diagnosis is to be made we must determine upon what the form and width of the palpebral fissure depends under physiological conditions.

I. Form and Width of the Palpebral Fissure under Physiological Conditions

We may call the form of the palpebral fissure in man almond shaped, in contrast with that of many animals, in which it is round. Its transverse form enables man to move his eyes in a large arc horizontally without causing the cornea to disappear. The length of the palpebral fissure of an adult man, measured from one canthus to the other, is about 30 mm, its width at the widest place, when normally opened, is from 11 to 14 mm. The eye of a child appears peculiarly wide open by comparison, because the longitudinal diameter is considerably less than in adults, while the vertical diameter is approximately the same. According to *Reuss* the palpebral fissure of a child two months old is about twice as long as wide, while in an adult the ratio is about as three to one. The apparent size of a child's eye depends moreover on the fact that usually only a little of the sclera is visible, so that the palpebral fissure seems to be almost filled by the cornea. The upper lid commonly covers the uppermost segment of the cornea, while the edge of the lower lid touches its lower margin. We may open our eyes widely, or we may squeeze the lids together, and between these two extremes are all possible degrees in the width of the palpebral fissure. This motility of the lids is due primarily to the activity of the levator and orbicularis, the former supplied by the oculomotor nerve, the latter by the facial, with the assistance of the muscles supplied by the sympathetic, the palpebralis superior and inferior and the orbitalis.

The palpebralis superior forms the middle layer of the aponeurosis of the levator and is inserted into the anterior margin of the tarsus. The fibres of the palpebralis inferior run in the middle portion of the lid to the lower, outer margin of the tarsus.

All of these muscles must work together in harmony and we can say that the width of the palpebral fissure depends on the proportional tonicity of the muscular tissue to the weight of the lids. The antagonism between the levator and the orbicularis, especially the tonicity of the levator, provides for its ordinary width. In weariness and sleep the tension of the levator yields and the upper lid sinks of its own weight over the eye. But the state of the palpebral fissure depends upon still other factors. The extrinsic muscles of the eyes have to preserve the equilibrium of the eyeballs, and the connections of these muscles with the periorbita, by means of the fascia, play a great part in this function. The eye is prevented from being drawn into the orbit by the tonicity of the four recti by these bands of fascia, while the action of the two obliqui is to draw the eye out from the orbit.

How much the width of the palpebral fissure can be influenced by a disturbance of these conditions of insertion and connection may be seen in this case of tenotomy of the internus; it seems to be somewhat enlarged, because the eyeball is pressed forward against the lids. Conversely we occasionally see a considerable narrowing of the palpebral fissure after an advancement.

Another factor of essential influence is the relative fullness of the orbit. The eyes are very prominent in many persons. The exophthalmos of *Basedow's* disease is caused by the relaxation and fullness of the intraorbital bloodvessels. The palpebral fissure is widened when the eyeball is pressed into it by retrobulbar tumors and inflammatory infiltrations in the orbit. It is doubtful whether any widening can be produced by spasm of the orbicularis. The orbitalis, supplied by the sympathetic, lies in man in the median portion of the fibrous membrane that covers the fissura orbitalis inferior, but it is very questionable if the contents of the orbit can be driven forward by the contraction of its scanty fibres. The size of the eyeball itself is of much more importance in this regard. I need only mention the protrusion of the long, highly myopic eye. The width of the fissure is also influenced by a number of reflex actions, such as the closure of the eyes when dazzled. It is constantly being affected by psychic emotions; it widens in fright and astonishment, it contracts, and the eyebrows are drawn together, in anger and hate.

The dependence of the width of the palpebral fissure on sensory irritations leads us into the field of pathology. The eye closes when irritated by a foreign body on the conjunctiva, and in inflammations of the cornea. It scarcely needs to be mentioned that the width of the fissure is affected by adhesions of the lids (ankyloblepharon), and cicatricial adhesions of the conjunctiva of the lids to that of the eyeball (symblepharon). Congenital blepharophimosis is occasionally met with.

After death the palpebral fissure usually remains half open, although it would naturally be supposed that a complete ptosis would follow relaxation of the levator and orbicularis. Probably the action of the orbicularis ceases later than that of the other muscles, and the quickly appearing rigidity causes this post mortem condition. During life we find the narrowest palpebral fissure associated with paresis of the levator. In this patient the cause of the narrowing is a paresis of the sympathetic, and we will pass to the analysis of the oculopupillary symptoms of this condition.

II. Analysis of the Oculopupillary Symptoms in Paresis of the Sympathetic Nerve

The palpebral fissure is rather narrower than the other, because the palpebralis superior, which is supplied by the sympathetic, is paretic, and its tonicity is overcome by that of the orbicularis, which is not paretic. In addition the upper lid sinks down a little from its own weight and the eyeball sinks back at the same time because of the paresis of *Mueller's* orbital muscle. The action of the palpebralis inferior is absent in the lower lid, so the tonicity of the orbicularis meets with no opposition and raises the lid a little.

The pupil is contracted because the tension of the sphincter pupillæ, supplied by the oculomotor nerve, preponderates when the dilatator no longer acts. I wish to call attention to the fact that this difference in the pupils is particularly distinct in reduced light. The pupil of the paretic eye does not dilate so promptly when shaded. The redness of the skin of the face is explained at once as due to the paresis of the sympathetic, but it is present only when the paresis is fresh, at a later period the paretic side becomes pale and no longer perspires.

The symptoms of stimulation of the sympathetic are exactly the opposite. When the principal trunk of the nerve is stimulated the pupil dilates and the side of the face affected becomes pale with an increase of perspiration. The palpebral fissure becomes wider because the lids are drawn back a little by the action of the palpebral muscles while the eyeball is made slightly exophthalmic by the contraction of the smooth fibres of the orbitalis.

III. Diagnostic Importance and Etiology of Paresis of the Sympathetic Nerve

The ocular symptoms produced by paresis of the sympathetic have a considerable importance in many cases. It has been known for a long time that tumors situated in the lateral portions of the neck, or in the upper part of the thorax, can produce this trouble in the sympathetic. Enlargement of the thyreoid gland is the most common, so we look first for signs of goitre. More rarely an aneurysm of the carotid may be the cause. Injuries of the neck, as well as tumors in the posterior mediastinum and in the upper part of the chest, must also be taken into account. We sometimes meet with it, though not, as a rule, simultaneously with traumatic pareses of the brachial plexus. The paresis of the lower plexus is known as *Klumpke's* paresis. *Frau Klumpke* has shown experimentally that narrowing of the palpebral fissure, meiosis and exophthalmos appear after section of the eighth cervical, and especially of the first dorsal nerve. According to *Kraus* the oculopupillary symptoms are particularly to be localized in the first dorsal segment.

Syringomyelia is the disease of the cerebrospinal system in which these oculopupillary symptoms are met with. According to *Schlesinger* the paresis of the sympathetic is usually on the same side on which the muscular atrophies are distinct. It forms an important initial symptom of the cervical form of this disease.

A differential diagnosis of this condition can be made by the instillation of a few drops of a 1% solution of cocain into the conjunctival sac; the pupil cannot dilate if a paresis of the sympathetic is present.

155. Paresis of the Facial Nerve and Keratitis e Lagophthalmo

The history of this patient is as follows: E. B., 19 years old, parents and living brothers and sisters healthy, five brothers and sisters dead from unknown causes. No family history of nervous or mental disease, or of convulsions. Development normal, well educated. He is said to have had at the age of 6 a paresis of the right side of the face which the physician thought might be due to an apoplectic attack. Otherwise he has always been well, he denies infection, smokes and drinks moderately. About the middle of last January he noticed noises in both ears and an impaired power to discern spices when eating, although he does not remember that he had a cold, or that he had been exposed to a low temperature. Both symptoms disappeared at the end of the day. A few days later he noticed that his right eye lacrymated, and within three days that the left side of his face was drawn to the left. He was then treated with electricity and medicine. The right side of his face twitched at first, but this ceased later. Speech was not impaired, there was no paræsthesia or weakness in any limb.

Condition: Strongly built, well nourished man with strongly developed muscles. Cranial nerves sensitive to tapping and pressure. Measurements of the head, from occipital protuberance to root of the nose, 33 cm; from one ear to the other, 30 cm; circumference, 54 cm. Eyebrows united by a bridge of hair. Pupils of medium size, round, react well to light and convergence. Movements of eyes free; some twitching like nystagmus on looking to the right or the left, which quickly ceases; corneal reflex present. Place of exit of fifth cranial nerve not sensitive to pressure. The left half of the forehead alone is wrinkled by the corrugator supercilii. When the eyes are closed a broad white stripe of the right eyeball is left uncovered by the lids. The mouth is drawn to the left; its left side alone moves in showing the teeth and in speaking. The tongue is protruded straight forward, is not tremulous and the pharyngeal reflex is weak. The peripheral arteries are soft and not tortuous. The pulse is regular, 80 beats per minute. The hands when outstretched do not tremble, the body does not waver when the feet are close together and the eyes closed, he stands securely on either leg alone. Patellar reflexes not increased, no distinct difference between them, though perhaps the right is a little the stronger, Achilles reflex present, no *Babinski*, no *Oppenheim*, cremasteric reflex positive, abdominal reflex present and symmetrical arm reflex not increased. The great nerve trunks are not sensitive to pressure, strength is well preserved, dynamometer pressure: right, 75, 75, 80, 80 = 320; left, 80, 80, 85, 78 = 323. No points painful on pressure, slight touches felt everywhere, sharp and blunt distinguished at all points. Needle pricks felt uniformly on both sides, cold and heat recognized well everywhere. Internal organs without demonstrable morbid conditions. No albumin or sugar in the urine. Sense of smell good on both sides. Taste better on the left side of the tongue than on the right. The right facial nerve *does not respond to faradism.* Galvanism of the branch to the mouth causes slow contraction, K. C. C. < A. C. C.; of the branch to the right brow and eye causes quick contraction, but rather slower than on the left, K. C. C. > A. C. C. The excitability is reduced. Hearing is intact; the secretion of saliva is not abnormally increased; the velum is raised evenly. Nothing abnormal to be seen in the fundus. Psychic condition normal. The treatment of this patient is galvanism daily with exercise and massage of the facial muscles.

This is a cases of paresis of one facial nerve, and we have three duties to perform: 1, To analyze the ocular symptoms accurately; 2, to try to locate the lesion; 3, to consider the therapeutic measures necessary to be taken in regard to the eye in every case of facial paresis.

I. Analysis of the Ocular Symptoms in Facial Paresis

In a case of paresis of one facial nerve the forehead on the affected side is smooth and not wrinkled because the frontal portion of the occipitofrontalis is paretic. The opposite eyebrow may be either higher or lower than the other, as it is drawn downward by the tonicity of the orbicularis, or upward by the contraction of the frontal portion of the occipitofrontalis. The most important phenomenon is the gaping of the palpebral fissure, known as lagophthalmos. It is very distinct in this patient in whom the edge of the lower lid is several millimetres below the margin of the cornea. This lagophthalmos is produced in the following way. When the orbicularis is paretic the weight of the upper lid is the only factor that acts to narrow the palpebral fissure, but this effect is counteracted by the levator and palpebralis superior, which are not paretic, so that at first the upper lid is higher than that of the healthy eye. This retracting action of the levator varies greatly in different cases of facial palsy. When the action of the lower part of the orbicularis ceases the lower lid is no longer pressed by it against the eyeball, while the tonicity of the palpebral muscle unites with its weight to cause it to sink, and a paralytic ectropion is produced. When the patient closes his sound eye tightly the skin of the brow and of the vicinity of the eye on the paretic side is drawn over toward the inner canthus of the opposite eye.

The next most important symptom is the absence of voluntary and reflex closure of the lids. If the physiological reflex action of winking is absent the cornea can no longer be kept moist and the bits of dust that fly into the eye can no longer be removed. The lacrymal gland responds to the irritation produced in this way with an increased secretion of tears, and this lacrymation is increased still more by the ectropion and the eversion of the lacrymal puncta. The tears are no longer driven toward the inner canthus as they are under normal conditions, but flow over the margin of the lid because they cannot easily enter the puncta which are not held in their proper positions by the palpebral fibres of the paretic orbicularis. This increase in the secretion of the tears keeps the cornea moist during the daytime, as the fluid is distributed by the movements of the eye in the place of winking, but during sleep these movements of the eye cease and the secretion of tears lessens on account of the diminution of the tactile irritation of the cornea and conjunctiva. Hence the cornea is in danger of becoming dry during sleep. This danger is often lessened for a while in young persons by the normal elasticity of the skin which presses the lid upon the eyeball, but in older persons, whose skin has become relaxed, or after the paresis has lasted a long time, changes take place in the conjunctiva and cornea, as may be seen in this patient. The conjunctiva in the vicinity of the lower margin of the cornea becomes red and a tenacious mucus accumulates at this place which dries and forms thick crusts on the cornea. Finally a keratitis e lagophthalmo develops. The occurrence of the complication is favored by sleep, the age of the patient and the long continuance of the paresis.

II. Localization of the Facial Paresis

Every case of facial paresis is of diagnostic and therapeutic interest to the physician. An attempt should always be made to locate the site of the lesion, which may vary a great deal. We will first refresh our memories in regard to the course of the facial nerve, especially of the branch that supplies the eye.

Course of the Ocular Branch of the Facial Nerve

The cortical centre of the upper facial nerve is in the lower third of the anterior central convolution. A number of investigators are of the opinion that the cortical centre of the upper does not coincide with that of the lower facial nerve, which is placed by *Monakow* in the second frontal convolution, by *Mendel* in the gyrus parietalis inferior. At all events the nerve tracts of the facial converge from the centre in the cortex through the centrum semiovale to the internal capsule. Behind them are the tracts for the arm and the leg. From the inner knee of the internal capsule we follow the tract of the facial into the pes pedunculi. The fibres lie separated from the pyramidal tract in the pons and reach the facial nucleus of the other side in a way that has not yet been sufficiently elucidated. Probably the upper facial occupies a tract distinct from that of the lower in the peduncle, for we find the lower facial alone involved in circumscribed diseases of the peduncle which give rise to hemiplegia alternans, oculomotor paresis with paresis of the limbs on the opposite side, while if the whole peduncle is destroyed the facial paralysis is total.

The nucleus of the facial nerve lies in the posterior part of the dorsal half of the pons, about 4½ mm to the ventral side of the floor of the fourth ventricle. According to *Koellicker* its entire extent is about 3½ mm. The connections of the facial nucleus with other nerves are of clinical importance, but I will mention only its relations to the trigeminus, which we need to know in order to understand the reflex symptoms. According to *Koellicker* fibres extend from the ventral portion of the sensory roots of the trigeminus to the nucleus of the facialis.

When the facial nerve leaves the brain it bends forward and to the side in company with the auditory nerve and with it enters the meatus auditorius internus. At the bottom of this meatus it passes through an opening into the Fallopian canal, in which it runs forward and then bends backward and to the side at a right angle, runs in the median wall of the tympanum over the fenestra ovalis at first backward, then downward and comes out through the stylomastoid foramen. From this point the nerve passes obliquely downward and forward and divides into its two principal branches behind the ramus of the lower jaw. The upper branch, the temporofacial, in which we are specially interested, passes through the parotid gland. Its motor twigs extend from the anterior muscles of the ear to the upper lip. The upper part of the orbicularis is supplied by the temporal nerve, its lower part by the malar.

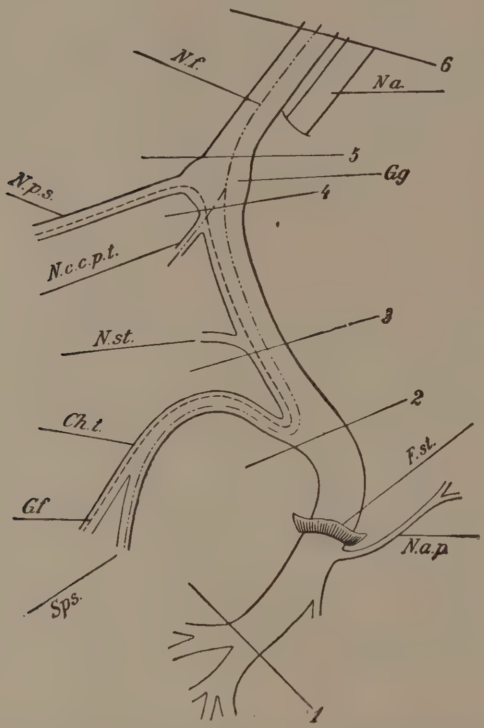


FIG. 124.—Schematic Diagram of the Trunk of the Facial Nerve from the Base of the Skull to the Pes Anserinus.

Nervus Facialis = *N. f.* Nervus Petrosus Superficialis Major = *N.p.s.* Nervus Communicans cum Plexui Tympani = *N.c.c.p.t.* Nervus Stapedius = *N.st.* Chorda Tympani = *Ch.t.* Gustatory fibres = *Gf.* Nerve of the Salivary Secretion = *Sps.* Nervus Acusticus = *N.a.* Ganglion Geniculi = *G.g.* Foramen Stylomastoideum = *F.st.* Nervus Auricularis Posterior = *N.a.p.* *Erb's* Diagram from *Struempell's* Textbook.

Localization of a Facial Paresis

When we wish to diagnose the site of the lesion in any case of facial paresis the condition of the orbicularis furnishes important data, for the rule generally holds good that the twigs which supply the mouth are the only ones permanently affected in cortical and subcortical affections of the facial nerve. Paresis of the entire facial nerve is on the contrary usually caused by a nuclear, infranuclear, or peripheral lesion. The differentiation must be made by the electric tests in doubtful cases. In the true cerebral pareses the electric excitability is perfectly preserved. We find reactions of degeneration only in peripheral pareses and those in which the lesion is situated below or in the nucleus. By this division into partial and total facial paresis we obtain a diagnostic point. The branches of the facial to the eye are found to be injured only in rare cases of cortical foci, such as softenings and tumors.

Most facial pareses of nuclear origin are bilateral, because of the neighboring position of the nuclei. In tabes, in which the other nuclei of the cerebral nerves are apt to be diseased, facial paresis is comparatively very rare. The nuclear affection of the facial is more frequent in acute polioencephalitis superior hæmorrhagica, according to *Wernicke.* A special description is needed only for the facial pareses met with in diseases of the pons.

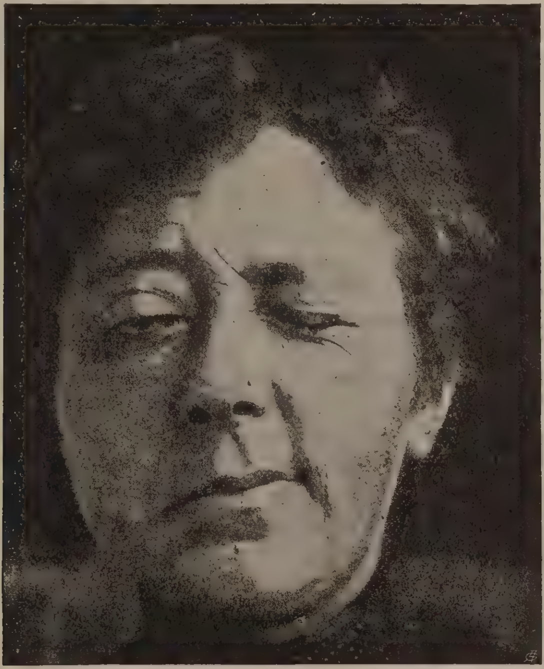


Fig. 125.—Right Facial Paresis Due to a Sarcoma of the Mastoid. Taken at the Moment of a Spasmodic Closure of the Lids.

The paresis characteristic of disease of the pons is hemiplegia alternans, paresis of the facial on the side corresponding to the seat of the disease and of the limbs on the opposite side. This is present only when the lesion is situated in one side of the pons above the decussation of the pyramids and below that of the facial nerve. If the lesion is above the decussation of the facial the paresis both of it and of the limbs is on the opposite side of the body. This paresis, known as *Gubler's,* involves all the branches of the nerve.

Peripheral Facial Paresis

The diagnosis of a peripheral facial paresis, in which usually all the branches of the nerve are involved, is very easy. The forehead is smooth and unwrinkled, the eye is wide open, the mouth is drawn to the healthy side, the nasolabial fold is smoothed out. *Erb's* schematic diagram (Fig. 124) is of service in determining the site of the lesion. The principal types are: 1. Paresis of the muscles of the face, leaving the secretion of saliva, the taste, the hearing and the velum intact; site of the lesion between the lines 1 and 2. 2. Paresis of the muscles of the face, impairment of taste, and diminution of the secretion of saliva, with hearing and velum normal; site of lesion in the aquæductus Fallopii. 3. Paresis of the muscles of the face, impairment of taste, diminution of the secretion of saliva, abnormally fine hearing, velum normal; site of lesion between lines 3 and 4. 4. Paresis of the muscles of the face, diminution of the salivary secretion, impairment of taste, acute hearing and paresis of the velum; site in the lesion in the ganglion geniculi, between 4 and 5. 5. Paresis of the muscles of the face, diminution of the salivary secretion, acute hearing, paresis of the velum, but no disturbance of taste; lesion above the ganglion geniculi, between 5 and 6.

The causes of peripheral facial pareses are first, exposure to cold, when we call it rheumatic. *Minkowski* has shown that this is a degenerative, rather than an inflammatory neuritis. Second, diseases of the middle ear and caries of the petrous portion of the temporal bone. It can easily be understood how the facial nerve in its course through the Fallopian canal in the immediate vicinity of the tympanum may become involved in such inflammations, or compressed by exudates. We primarily examine the ears in all such cases. Lesions at the base of the skull, such as fractures, aneurysms, tumors, acute and chronic meningitis, must also be taken into account. Other nerves, like the acusticus, abducens and trigeminus, are usually involved, but the gustatory fibres frequently are not affected in facial pareses of basal origin. Tumors and injuries of the parotid and of its vicinity may also cause a peripheral facial paresis.

As regards functional pareses of the facial nerve it is of diagnostic interest to know that the lower branch is the one always affected in a hysterical paresis, while that to the eye is spared. Facial paresis never occurs in epilepsy, but it has been observed in rare cases of hemicrania and may even recur periodically.

This gives an idea at least how we try to locate the lesion in a case of facial paresis. The treatment to be adopted depends on the etiology. The course when due to disease of the middle ear depends on the curability of the latter. Aspirin, electricity and massage are our best remedies in rheumatic pareses. If the electric excitability remains normal during the first weeks the prognosis is good, but other rheumatic cases require months for recovery, or may be incurable. The latter is naturally the case when the paresis is caused by a tumor.

III. Prophylaxis and Treatment of Keratitis e Lagophthalmo

No matter where the lesion may be situated the physician has an important therapeutic duty in every case of facial paresis. The most important symptom, so far as the eye is concerned, is the lagophthalmos, the incomplete closure of the eye. When the paresis is slight the palpebral fissure may be closed to some degree by a strong effort, but during sleep, when the tonicity of the orbicularis is relaxed and the eyelids close gently, patients with this trouble lie with half open eyes. This condition was named hare's eye (lagophthalmos) because hares were once believed to sleep with their eyes open. The imperfect covering of the eye leads to trouble which it is the duty of the physician to prevent. When our patient with facial paresis closes his lids the eye can be seen to turn upward, so that a great part of the cornea is protected by the upper lid, but its lower margin and the adjacent conjunctiva scleræ remain exposed to the air. It is rare that a lagophthalmos is of such high degree that the cornea remains completely uncovered. The conjunctiva exposed to the action of the air responds with hyperæmia and secretion, and the patient has a severe conjunctival catarrh. The cornea suffers still more; the place left bare by the lids becomes dry and a disease is started which is called keratitis e lagophthalmo. The dry epithelium becomes cracked and is cast off, and then inflammatory agents, of which there is no lack in the conjunctival sac, enter the parenchyma of the cornea, which becomes cloudy at this place, the superficial layers break down and an ulcer is formed. In the most fortunate cases the ulcer heals, leaving a small leucoma, in others it finally results in perforation. One characteristic of this condition is that the dryness always affects the lower part of the cornea.

In other cases no ulceration takes place, but the epithelium becomes thickened so as to resemble epidermis. This sort of xerosis renders the surrounding cornea more or less opaque.

The cause of keratitis e lagophthalmo is not always a facial paresis. Imperfect closure of the lids may be due to mechanical obstacles, as in exophthalmos from various causes, or in the shortening of a lid as the result of injuries, operations, or tumors. Such a dryness of the cornea is not infrequent in unconscious patients whose eyes close imperfectly, due to the loss of the lacrymal secretion during the somnolent condition. The opacities left in the lower segment of the cornea by a keratitis e lagophthalmo often indicate in later years the serious disease to which it has been subjected. Recently a man came into the clinic who had had several years before a very severe attack of typhoid fever with disordered consciousness, and presented the peculiar opacities at the margin of the cornea from which we could diagnose the former existence of a keratitis e lagophthalmo. Whatever may be the cause of this disease in any given case we must try to prevent its development from the start.

During the day the feeling of dryness usually suffices to excite an increased reflex action of winking: if this is absent or impaired we must bandage the eye. This must always be done during the night. A large quantity of some indifferent ointment, like that made with boric acid, is applied to the open palpebral fissure, and over it is secured an ordinary moist dressing. The original cause of the lagophthalmos must be dealt with. An ectropion, or a shortening of the lid, must be obviated. Cases are met with in which the eyelids are congenitally short; such persons sleep with imperfectly closed eyes and develop, as a rule, an obstinate blepharitis, which must not be thought to be the primary cause of the lagophthalmos. If the fundamental cause cannot be obviated and the lagophthalmos is incurable we try to shorten the palpebral fissure so as to prevent as far as possible the danger of desiccation of the cornea. We employ the little operation of tarsorrhaphy for this purpose.

156. Prognosis and Treatment of Pareses of the Ocular Muscles

We have found a great variety both in the causes of pareses of the ocular muscles and in the sites of the lesions that produce them, but our duty is not done with the clinical analysis. We are not simply diagnosticians, we are physicians as well. We must not only determine which muscle is affected and try to locate the lesion, but we must endeavor to ascertain its true cause in order to attack it with our therapeutic measures. Two ways are at our disposal by means of which to determine the cause. Here, in the clinic, where it is my duty to make my students acquainted with the eye symptoms, we naturally try first

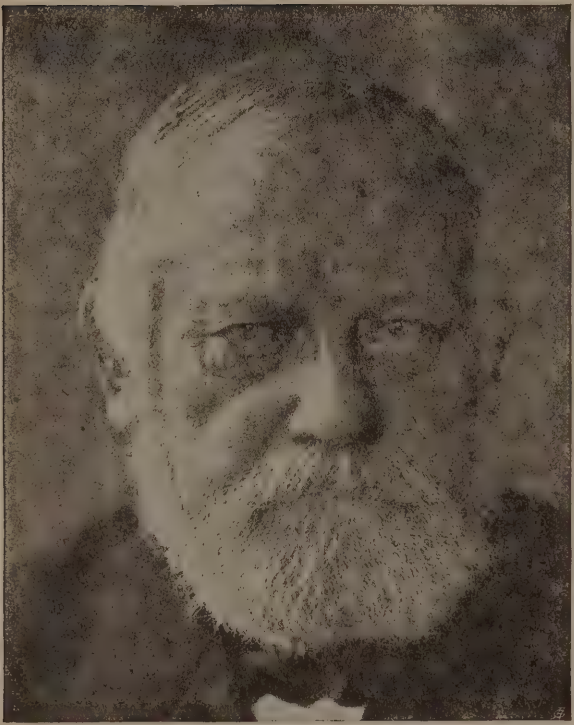


FIG. 126a.—Primary Position.

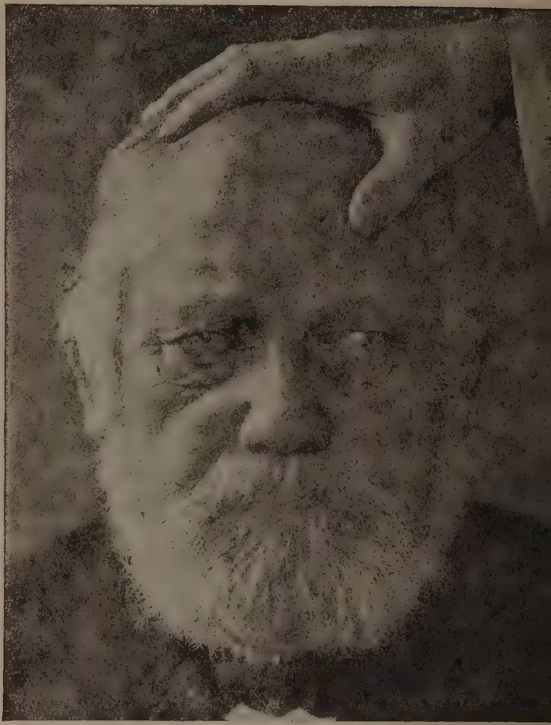


FIG. 126b.—Paresis of the Right Internal Rectus.

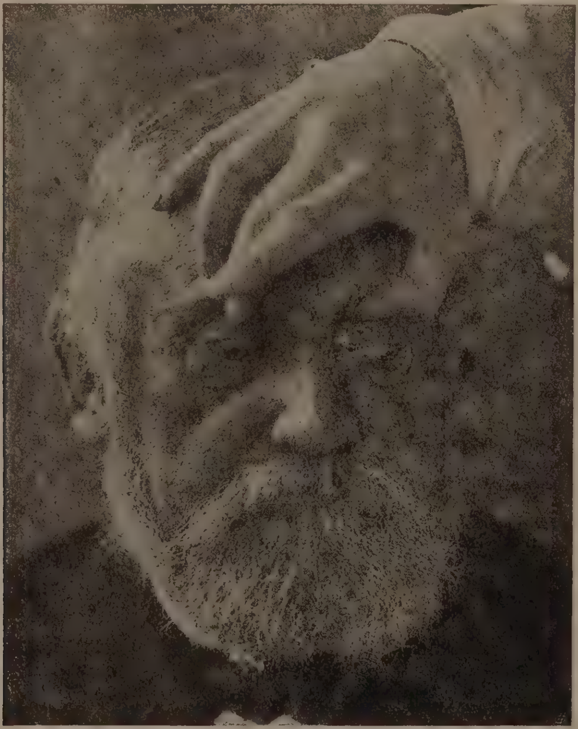


FIG. 126c.—Paresis of the Right Trochlearis.

Paresis of Right Internal Rectus and Trochlearis in a Case of Arteriosclerosis.

to read from these the cause of any paresis that may be present. This is the reason why I have arranged such practices in differential diagnosis as we have had in connection with the ophthalmoplegias and with ptosis. But we must be honest enough to admit that such a confinement to the eye symptoms leads by no means always to the desired result. The eye can tell us much, but often not all. We need the examination of the entire organism from the point of view of the internist, and the neurologist, to discover the etiology of a paresis of an ocular muscle. Take, for example, a case of ophthalmoplegia interna of one eye. We know that syphilis is the first thing to be thought of, but we must have certainty, rather than probability, and therefore have to call in the aid of *Wassermann's* reaction. Every paresis of an ocular muscle should cause the physician to make a thorough examination of the entire organism. With this monition firmly fixed in mind our exercises in the diagnosis of these cases will not have been in vain. We undertake this general examination and carry it as far as we are able, but it is the rule in this clinic that every neurologic eye case is reexamined by either the internist or the neurologist. I am particularly grateful for the cooperation of my colleague *Schultze* of the neurological clinic. How much the cause of a paresis of an ocular muscle may vary can be inferred from what we have learned in our study of the differential diagnosis of ptosis. From

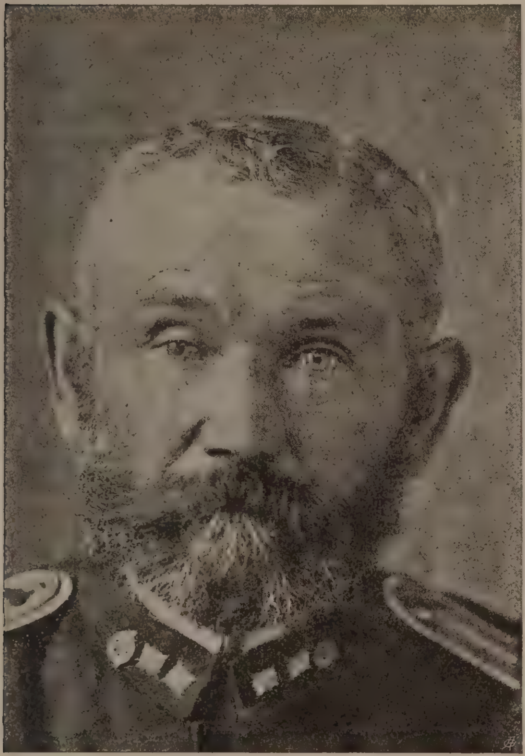


FIG. 127a.—Primary Position: The Left Eye Is Fixing, the Right Is Deviated Downward by the Secondary Contracture.

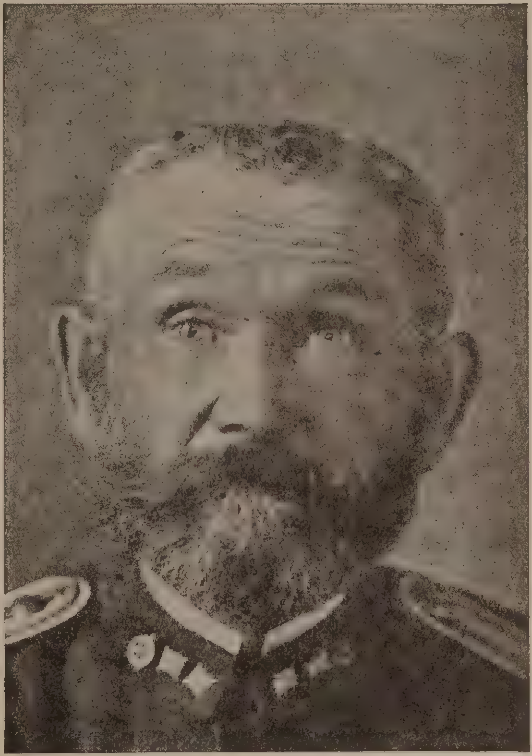


FIG. 127b.—Lagging Behind of the Right Eye in Looking Upward, the Left Eye Still Fixing.

Old Paresis of the Right Superior Rectus.

these differences in the etiology we obtain two clinical points with which we may bring this discussion to a close, the prognosis and the treatment.

I. Prognosis of Pareses of the Ocular Muscles

It is evident that the prognosis varies in the individual case according to the site of the lesion, as well as the extent, severity, and nature of the disease, so it is impossible to state anything positively. In general it may be said that the prognosis of rheumatic pareses, as well as of those due to slight intoxications or infections is good. Likewise those due to minute hemorrhages in the region of the nuclei are accustomed to improve more or less. I have already stated that tabetic pareses have a fleeting character, they disappear quickly, but also reappear. The result in syphilitic pareses cannot be calculated upon; sometimes they disappear promptly under specific treatment, but often they are not influenced at all. I can demonstrate this by a case which has been under the joint treatment of this and the neurological clinic.

P. Oskar, locomotive engineer, 54 years old. According to the statement of his physician he contracted syphilis six years ago and was placed at once under treatment, but discontinued this after ten injections. About three months ago he noticed that he raised his eyes with difficulty, and then he began to see double.

A strong muscular tremor can be seen in his right upper lid when his eyes are closed. When he looks at a distance his right eye deviates 25° outward and 5° upward. An interpretation of the double images is possible only in so far as the outward deviation may be ascribed to a marked paresis of the internal rectus together with a slight paresis of the superior rectus. As the right eye maintains common fixation with the left for only a short time, in consequence of the paresis of the internus which has lasted some months, nystagmoid twitchings of both eyes, but especially of the right, follow every direction to fix, after which he usually succeeds in fixing an object binocularly, even though it is only 15 cm distant. The right eye then deviates outward very quickly and occasionally the left internus appears to be weak. In addition to this every movement of the eye is visibly associated with a certain strain, even when he looks in directions in which the eye still maintains maximal excursions. The symptom of a bilateral exophthalmos is explainable in part perhaps by a general relative paresis. It is greater in the more paretic eye and is, as measured with *Hertel's* instrument, right, 22 mm; left, 18 mm. It is difficult to determine whether there is any paresis of the accommodation or not, but as, being a myope of 1 dioptre, he should at his age read print at 50 cm, and as he reads the finest print at 25 cm with 2 D, the accommodation cannot be greatly impaired. On the other hand both pupils are dilated and have reflex immobility.

The neurological report is briefly: Small, corpulent man with strong muscles. Fifth nerve intact. Seventh nerve symmetrically hyperinnervated, especially the lower branch. The tongue deviates to the left when protruded, trembles and undulates violently; the lower jaw also trembles. Considerable tremor of the hands, no *Quinquaud*, strength good, no ataxia. Sensibility to touch and pain good. Hyperæsthesia for cold in the groins. Movements in the small joints normal. Patellar reflex weak. Right Achilles reflex weaker than the left. Cremasteric reflex +. Abdominal reflex lively. Arm tendon reflex +. Corneal reflex +. Psychic condition: Dull sense of comfort; always replies that he feels well. Power of observation much impaired. Apprehension dulled. Serious impairment of speech.

II. Treatment of Pareses of the Ocular Muscles

In such a case as this the treatment is naturally directed toward the cause, mercury and iodide. In rheumatic pareses we use aspirin, sweating and hot baths. When the case is one of arteriosclerosis, the treatment must be in this direction.

Local, symptomatic treatment is to be added. Galvanization with weak currents is the principal remedy. The kathode is placed on the nape of the neck, the anode on the lids or temple. The effect produced is often good for a time, but the final result is apt to be slight. We may be of assistance in one way in many cases. The symptom which annoys the patient most is the diplopia, and this disappears at once as soon as one eye is covered. The patient does this for himself in case of need by closing one of his eyes. We may therefore cover one with a patch, or exclude it from the act of vision by means of an opaque glass. The paretic eye is the one usually excluded in this way. Treatment with prisms is useful to only a very limited extent.

Operative treatment is to be thought of only in suitable cases, after the disease has passed completely away and a large degree of paralytic strabismus persists after months of treatment. One indication for an operation is when we can establish the fact that the strabismus is increasing through secondary contractures of the antagonistic muscles. In such a case we first try advancement of the paretic muscle, with tenotomy of its antagonist when its secondary contracture is abnormally high. Operative intervention is particularly justifiable in paresis of one of the obliqui. Advancement of the antagonist, with perhaps tenotomy of the rectus of the other eye that acts in concert with the paretic muscle, may be indicated in such a case. For example, if the left superior oblique is paretic, so that the eye is deviated somewhat upward and inward while its upper pole is inclined to the left, we may tenotomize the inferior rectus of the right eye so as to deviate the line of vision of the right eye upward. At the same time a diminution of the convergence may be secured, and the corresponding meridians rendered more parallel in this way, for by weakening an outward rotator of the right eye its upper pole must be inclined to the left, the same as that of the paretic eye. All operative interventions are contraindicated when complicated pareses of the ocular muscles are present.

XV

Neurology of the Eye

157. The Relations of the Trigeminus to the Eye, and Trigeminal Neuralgia

This patient tells us that very severe pains radiate at times from a certain point above his right eye, which corresponds to the supraorbital foramen over his forehead. Pressure on the supraorbital nerve causes him to shrink from the pain, and he seems to have a neuralgia of this branch of the trigeminus. We have a great deal to do with the irritations and diseases of this nerve in the eye clinic, so we will first recapitulate the portion of its anatomy which is of clinical interest to us. The first question is: How do the fibres of the trigeminus reach the eye?

1. Course of the Trigeminus to the Eye

The trigeminus arises by two roots at the base of the brain from the pons, or the ventral surface of the middle crus cerebri. The anterior root contains the motor, the posterior the sensory fibres. They lie close together with the motor root on the lower median side of the other. The nerve passes over the apex of the petrous portion of the temporal bone through an aperture in the dura a little to the side of the place of exit of the abducens, into a cavity formed by the upper surface of the petrous portion of the temporal bone and the dura, known as the cavum *Meckelii.* In this cavity, which is situated laterally from the sinus cavernosus and extends forward to the median angle of the fissura orbitalis superior, the foramen rotundum and the foramen ovale, the posterior root forms the *Gasserian* ganglion. Beneath passes the anterior motor root to unite with the third of the three branches of the trigeminus that arise from this ganglion. The first of these branches, the ramus ophthalmicus, enters the orbit through the superior orbital fissure, in which it divides into three principal branches, the supraorbital, lacrymal, and nasociliary nerves. These three branches supply, through their filaments, the skin of the head from the palpebral fissure to the vertex of the skull, the conjunctiva, cornea, iris, ciliary body and part of the nose. The twigs of the supraorbital nerve send sensory fibres to the upper lid, eyebrow, glabella and the skin of the forehead and temple as far up as the vertex. The lacrymal sends sensory twigs to the conjunctiva, upper lid and the skin of the temple, and also furnishes secretory fibres to the lacrymal gland. The nasociliary nerve supplies the conjunctiva, caruncle, lacrymal sac, upper lid, eyebrow and root of the nose; one of its branches, the ethmoidal nerve, supplies the tip and a large part of the interior of the nose. From the nasociliary nerve the radix longa extends to the ciliary ganglion, and the long ciliary nerves pass directly to the eyeball. The ciliary ganglion lies in the orbit between the optic nerve and the external rectus. It has three roots, the radix longa or sensory, from the nasociliary nerve, the radix brevis, or motor, from the oculomotor nerve through a branch that runs to the inferior oblique muscle, and the radix sympathica, from the carotid plexus. The ciliary ganglion gives rise to the short ciliary nerves, which perforate the sclera near the entrance of the optic nerve together with the long ciliary nerves. These ciliary nerves pass forward between the sclera and chorioid, and form a large close meshed plexus in the ciliary body from which the iris and cornea are supplied. The nerves to the cornea pass into it from the sclera, together with minute ones from the conjunctiva.

2. The Physiological Relations of the Trigeminus to the Eye

It must be remembered that the ciliary nerves contain fibres not only of the trigeminus, but also of other nerves. They contain 1, the sensory nerves for the cornea, conjunctiva and iris, which excite in a reflex manner the flow of tears (the lacrymal nerve), and the closure of the lids (the facial nerve); 2, the vasomotor nerves for the vessels of the iris, chorioid and retina, that come primarily from the sympathetic; 3, the motor fibres for the dilatator pupillæ from the sympathetic root of the ciliary ganglion. We learn whether the physiological relations of the trigeminus to the eye are normal or not by ascertaining the quality of sensibility in the anterior segment of the eyeball.

We determine first whether the sensibility of the cornea and conjunctiva is normal by testing it with a bit of cotton. Views are still divided in regard to the qualities of sensation possessed by these tissues. *v. Frey* tested the sensitiveness of the cornea with hairs of different sizes, and according to *Nagel* the conjunctiva and cornea can differentiate between heat and cold, but the cold touch alone produces a specific sensation of temperature. At the same time that we test the sensitiveness we notice whether the reflex activity of the trigeminus to irritation of the anterior segment of the globe is normal. A transient irritation of the cornea and conjunctiva causes a marked hyperæmia of the conjunctival vessels, the ordinary closure of the lids is excited through the trigeminus, and the eye responds to the irritation produced by touching it with an increased secretion of tears. The patient also makes a movement of the head to escape the pain excited in the cornea. Still other reflexes may be excited through the trigeminus in rare cases. Thus when the conjunctiva is irritated a short interruption of the respiration, due to an inhibitive action of the laryngeal nerve, may occasionally be observed. Reflex swallowing or sneezing may sometimes be caused by instillations of drops into the conjunctival sac.

3. Neuralgia of the Trigeminus

Our patient is hypersensitive throughout the region supplied by the first branch of this nerve. We speak in general terms of a neuralgia when the pain is fairly severe, appears in paroxysms, and is along the course of a certain nerve. The diagnosis of a typical neuralgia of the trigeminal nerve is therefore not difficult. The symptom which is more or less common to all these neuralgias is that the pains appear either quite suddenly, or after certain premonitory signs, such as a feeling of chilliness, tingling and slightly painful sensations. The patients can specify accurately the extent of the pain, which corresponds to that of the nerve affected.

An objective examination reveals distinct disturbances of sensation. We test whether the sharp end of a needle can be distinguished from the blunt, whether the perception of heat is normal, etc. During the interval between attacks there is in many cases a more or less marked anæsthesia, but usually we find the affected area hyperæsthetic. We also look for certain points that are sensitive to pressure. When we press upon the place of exit of this nerve beneath the superciliary arch the severe pain causes the patient to groan. A distinctly increased sensitiveness to pressure may be found at this point even during the intervals. These sensitive points are generally to be found at places where we can press the nerve against a firm substance. Such a point usually exists at the supraorbital foramen in supraorbital neuralgia, less frequently at the inner canthus and on the tuber parietale. In neuralgia of the second branch we find them at the infraorbital foramen and on the malar bone. The alveolaris inferior is the portion of the third branch that is most commonly affected, though a neuralgia of the auriculotemporal, or of the lingual may occur. The principal point sensitive to pressure is at the mental foramen.

The course of trigeminal neuralgia varies a great deal. The pain may be mild and the attack pass off quickly, or it may be extreme and radiate into the neighboring parts, into the occiput, the nape of the neck and the shoulders. The attacks may be of very short duration, or they may last for hours. The intervals between the paroxysms likewise vary a great deal. At these times the patients may feel perfectly well, but a certain sensitiveness of the skin persists in many of them. Extremely severe, incurable cases have driven persons to suicide. All of this depends naturally on the cause and the severity of the disease. The neuralgias of the first branch of the trigeminus, with which we have mainly to deal, are usually mild. A physician often confines himself to the diagnosis merely of trigeminal neuralgia, based on the sensitiveness of the nerve to pressure, but he should in every case try to determine whether the neuralgia is primary, or secondary to some other disease. These affections occur most often in middle and old age, are met with more in women than in men, and are favored by a neuropathic predisposition, the bodily constitution, anæmia and overstraining.

The causes are:

1. The so called catching cold. We speak of these as cases of rheumatic neuralgia.
2. Injuries in which the nerve has been crushed.
3. Infections and intoxications. We meet with neuralgia of the trigeminus in herpes zoster, during the course of malaria, in the secondary stage of syphilis and in chronic alcoholism.
4. Constitutional anomalies. Neuralgias are not of rare occurrence in gout and diabetes.

We must take all of these things into account in each individual case, but quite a number of morbid changes occur in the vicinity of the eye, to which our attention is first attracted by the presence of a trigeminal neuralgia. We look immediately for disease of the teeth in neuralgia of the second branch, but in all forms we must ascertain whether the trouble is not caused by an affection of the nose, of the frontal sinus, or of the middle ear. A periostitis of the margin of the orbit may be the cause, and in many cases a trigeminal neuralgia is a sign, or a precursor, of an intracerebral disease.

The treatment naturally depends on the condition found to be present. Surgical intervention, neurectomy, becomes necessary in very severe cases, but frequently the causal indication is toward the extirpation of tumors, or the treatment of diseases of the accessory sinuses. Injections of alcohol along the course of the nerve have been made with success. A causal treatment can also be carried out in neuralgias that are due to such general diseases as anæmia and syphilis. Quinine and arsenic are indicated in malaria.

At the time of the paroxysm we may try such internal remedies as sodium salicylate, antipyrin, antifebrin, phenacetin and salipyrin. Locally we apply heat to the eye, use electricity and employ a careful massage.

158. Paresis of the Trigeminus and Keratitis Neuroparalytica

This is a case that I would like to impress firmly on the memory. The trouble seems to be corneal at first glance, and yet it appertains to the neurology of the eye, for the disease present is of a nerve. The patient presents a number of symptoms on the part of the central nervous system indicative of a tumor of the brain, into which we will not go in detail, but he would never have come to us if a very characteristic condition had not developed in one cornea a few days ago.

I. Clinical Picture of Keratitis Neuroparalytica

A fact which is very striking is that the eye is irritated very little, in spite of the gravity of the condition in the cornea. Ciliary injection is present, but the eye does not lacrymate, and the patient states that he has no pain. These two symptoms indicate a paresis of the trigeminus. The entire region supplied by the first branch of this nerve is anæsthetic. The picture presented by the cornea itself is quite peculiar; its centre is dull, opaque and denuded of epithelium far toward the periphery, leaving only a narrow strip at the margin which is clear and covered with epithelium. The floor of this large defect is cloudy.

In keratitis neuroparalytica the centre of the cornea first becomes dull, then the epithelium begins to be cast off; the cloudiness increases, the floor of the loss of substance becomes more and more gray, and may finally become purulent. Another striking point is that the course is extremely slow. In less severe cases the epithelium may remain in place, lustreless and dry. When it has once been cast off ulcerations readily develop from secondary affections, which at least leave behind them dense opacities, but usually go on to perforation, prolapse of the iris, or applanatio corneæ. As this disease is dependent on a paresis of the trigeminus, the prognosis naturally depends on the cause of the paresis. We are almost impotent in the presence of this inflammation of the cornea, and can treat it only in the same way as other forms of keratitis, with ointment, bandage, atropin and heat.

As neuroparalytic keratitis is of greater diagnostic than therapeutic interest I wish to call attention to the fact that it possesses a certain degree of similarity to keratitis e lagophthalmo and to keratomalacia, yet all three are easy to differentiate clinically. Keratitis e lagophthalmo begins, as a rule, in the lower segment of the cornea, in keratomalacia all parts of the cornea are dull and hazy. We meet with the latter chiefly in little children in whom the nutrition is greatly impaired, and we notice that the dryness persists even when the eye is bathed with tears, because the fluid cannot moisten the epithelium that has undergone fatty degeneration. In keratitis neuroparalytica on the contrary the secretion of the tears has been lessened because of the paresis of the trigeminus.

II. Origin of Keratitis Neuroparalytica

The study of neuroparalytic keratitis begins with the question whether the trigeminus contains trophic fibres, which pertain to the nutrition of the tissues in addition to the sensory ones.

Magendie showed experimentally that keratitis neuroparalytica appears in rabbits after intracranial section of the trigeminus, and ascribed it to the lesion of the trophic fibres which reach the eye through the ciliary nerves. Since his time numerous researches have been instituted, into the details of which we cannot go. I will simply mention the most important and sketch the development of our knowledge to its present condition.

The existence of trophic fibres in the nerve was contested very soon. *Snellen* showed that by suturing together the lids, or by suturing one of their ears into the palpebral fissures of the animals, the onset of a keratitis neuroparalytica was delayed, or wholly prevented. He believed this to be essentially a traumatic affection, due to the lodgment of foreign bodies on the cornea in consequence of the anæsthesia, which rendered the eye unable to protect itself any longer against very slight injuries. Other investigators made compromises. *Samuel* maintained that in a paresis of the trigeminus the power of resistance of the cornea was impaired in consequence of the lesion of the trophic nerves, so that insignificant injuries that would be borne by the healthy eye were productive of disease. Others, like *Schiff* and *Seydel*, thought more of the vasomotor influence. Section of the trigeminus causes a paresis of the nerves that supply the vessels, so they thought that vasomotor disturbances might be of importance, in addition to the anæsthesia, in the origin of this disease.

The contest wavered back and forth for a long time. The generally accepted opinion today is that two theories, the xerotic and the mycotic, are to be combined to explain its origin. *Feuer* showed by experiment that section of the trigeminus does not directly impair the nutrition of the cornea, but that the keratitis was rather due to the cessation of the act of winking. The portion of the cornea lying in the palpebral fissure becomes dry and the inflammation is introduced through this necrosis of the tissue. We can in fact essentially explain keratitis neuroparalytica by means of this drying of the cornea, as *v. Hippel* later set forth clearly, but for most cases the aid of the mycotic theory, propounded by *Eberth*, is also needed. *Eberth*, *Balogh*, *Panas* and many others are right in their claim that the entrance of inflammatory agents is made possible by this desiccation of the cornea. One thing at least has been decided, that true trophic nerve fibres are not present in the cornea, for *Krause* has performed quite a number of extirpations of the *Gasserian* ganglion on human beings without causing neuroparalytic keratitis.

I will repeat: We now think the cause of keratitis neuroparalytica to be that in consequence of the paresis of the trigeminus and the anæsthesia so produced the act of winking becomes more infrequent, the eye becomes less moist, the epithelium dries and necroses. In mild cases that may be all, or the necrotic tissue may excite a reactive infiltration of the cornea, but in grave cases microorganisms settle in the necrotic area and the inflammation extends. When there is no neuroparalytic keratitis in spite of a paresis of the trigeminus, conditions must be present which prevent the desiccation of the cornea or cause an increased amount of secretion. Such conditions are abundant. Some men wink more often than others. If a man who has been accustomed to wink less frequently suffers from a paresis of the trigeminus, the diminution in his winking and in the distribution of the tears over the eye may result in dryness of the cornea and keratitis, while in another, who winks more frequently, the same amount of diminution may not cause any noticeable disturbance. In addition to this there are other individual differences, such as those in the width of the palpebral fissure, and in the position of the eye in the orbit, which need to be taken into account. We must not forget that the power of resistance on the part of the epithelium of the cornea varies a great deal, as well as the predisposition of the eyes to the occurrence of secondary infections. I believe this to be at least a plausible explanation of how neuroparalytic keratitis is produced.

The next question is: What causes the paresis of the trigeminus?

III. The Causes of Paresis of the Trigeminus

It has been shown by the conditions found on autopsy in cases in which the trigeminus was diseased, collated by *Wilbrand* and *Saenger*, that paresis may be caused by a lesion at any point in the course of the nerve from the superior orbital fissure to the nuclear region.

The first question in the differential diagnosis of the site of the lesion is whether this is a total or a partial paralysis. A total paralysis of the trigeminus results not only in a sensory disturbance over the entire area supplied (face, conjunctiva, cornea, mucous membrane of the nose, mouth, tongue, cheeks and palate), but also in a paresis of the muscles of mastication, and sometimes in secretory troubles. Primary, isolated, total paralysis of the trigeminus is very rare. Total paralyses of this nerve are met with most frequently in connection with affections of the base of the skull, such as syphilitic meningitis, tumors, aneurysms, periostitis, hemorrhages, fractures, shot and stab wounds. A total paralysis must also be produced when the root of the trigeminus and the region of its nucleus in the pons is destroyed. Affections of the *Gasserian* ganglion also need to be taken into account.

If total paralysis of the trigeminus can be excluded in a case in which the condition present is one of anæsthesia of the area supplied by the first branch of this nerve, the interruption of the conductivity of which must render the conjunctiva and cornea anæsthetic, we must not think that the lesion is necessarily situated peripherally to the *Gasserian* ganglion. The cases of autopsy collated by *Wilbrand* and *Saenger* show that sensory disturbances may be excited in the first branch by lesions situated anywhere along the entire course of the nerve. The only thing necessary is that the fibres belonging to that branch be affected. The conductivity may be interrupted in the orbit, as by an opticociliary neurotomy, or by a gumma in the apex, or the lesion may be in the cavernous sinus, at the base of the brain, in the *Gasserian* ganglion, or even in the region of the root in the pons, but in general a lesion that injures a single branch causes sensory disturbances throughout the region supplied by the nerve. In trigeminal affections of the eye we frequently meet with sensory disturbances confined to small areas. The cornea and conjunctiva may be anæsthetic along with single patches of skin. Such a condition is indicative of a central lesion, according to neurological experience, because the region of the origin of the trigeminus is very large and may suffer partial destruction. We are indebted to *Oppenheim* for the proof of the important clinical fact that in tumors of the cerebellum an areflexia of the cornea and conjunctiva may exist for a long time. In many cases in which the trigeminus is affected the anæsthesia is confined to the cornea and conjunctiva. Such an anæsthesia may be produced by a peripheral lesion. A fact of practical importance is that this anæsthesia may be preceded by hyperæsthetic and neuralgic conditions when the cause is a growing tumor, or a meningitis. The sensory disturbance in the eye may also take the form of the so called anæsthesia dolorosa, when lively subjective neuralgic pains coexist with the objective absence of feeling. It is evident that in such cases the conductivity of the nerve is interrupted as far back as the lesion, to the central side of which it still responds to conditions of irritation.

After these rather general remarks concerning neuropathic keratitis we will consider what conditions have to be taken into account in practice in most cases.

1. Injuries of the Skull

Numerous cases have been reported after fractures of the base of the skull, shot and stab wounds.

2. Tumors of the Brain

Anæsthesia in the region supplied by the trigeminus is a frequent symptom of tumors of the cerebellum and especially of the pons. Solitary tubercles have a certain predilection for these situations. Neuralgic pains, paræsthesias and hyperæsthesias may precede the neuroparalytic keratitis in the case of a growing tumor.

3. Syphilis

When injury and tumor can be excluded syphilis is the probable cause, for the trigeminus is apt to be primarily involved in gummatous growths at the base of the brain, the same as the opticus, oculomotorius and abducens.

159. The Trigeminus and Herpes Zoster

There are some diseases of the eye the diagnosis of which is very easy. This is one of them.

After some days of lively neuralgic pain over the right eye an eruption of vesicles has appeared on the right side of the forehead and the right upper lid, especially in its inner portion. The upper lid is very œdematous and red, the swelling extends up to above the edge of the hair, but stops abruptly at the median line. The conjunctiva is much injected, the cornea is normal; the patient has photophobia and increased lacrymation, the preauricular gland is somewhat swollen. This is a herpes of the eyelid. Herpes appears on the eyelids in two distinct forms: as herpes simplex and as herpes zoster.

I. Herpes Simplex

Herpes simplex is a mild disease. A simple herpes appears on the lips in febrile diseases of the respiratory organs, and still more in digestive disturbances; associated with it single vesicles may appear on the eyelids, in the intermarginal space or at the outer canthus, or these may appear by themselves. The prognosis is good; the contents of the vesicles become cloudy and dry up after a few days. Treatment with a simple ointment is sufficient.

II. Herpes Zoster

Herpes zoster is distinguished from herpes simplex by two important features: the inflammatory symptoms of the skin disease itself are more severe and serious complications in the eye are much more common. We need to study the origin and course of herpes zoster in order to understand the clinical picture.

1. Origin and Course of Herpes Zoster on the Skin of the Lid

Herpes zoster ophthalmicus is an inflammatory affection of the skin, characterized by the appearance of vesicles or blebs along the terminal filaments of the trigeminus. This eruption of vesicles may take place on both the upper and lower lids, but usually the disease is confined to the ramification of the first branch of the trigeminus.

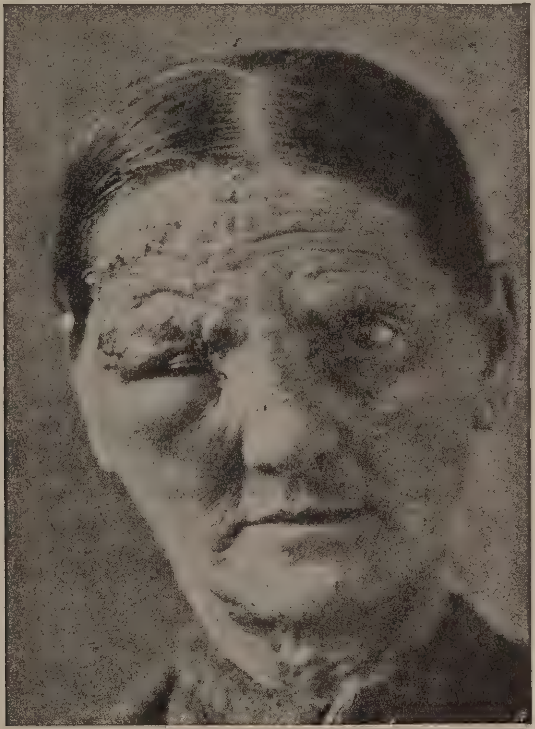


FIG. 128.—Right Eye: Herpes Zoster Ophthalmicus.

The eruption is usually preceded by neuralgic pains that are extremely severe and may outlast the vesicles. At the same time that the sensitiveness of the trigeminus is increased the eye itself begins to become photophobic, while the skin of the lid reddens and grows œdematous. The swelling may be so great that the upper lid can scarcely be raised. Then the vesicles appear; they may be quite discrete, may be confined to single twigs of the ramus ophthalmicus, or may be uniformly distributed. The inner third of the brow is the part affected, as a rule.

At this stage a careless observer may think of erysipelas, especially as the lymphatic glands of the ear are swollen, but the arrangement of the vesicles, the fact that the median line is rarely if ever passed over, and the neuralgic pains immediately indicate the diagnosis. All of the vesicles may be formed at once, but sometimes they appear in successive crops. Another clinically diagnostic sign is that in the overwhelming majority of cases herpes ophthalmicus affects only one side, as was pointed out by *Hutchinson*, though it may be bilateral at time. It may occur at any age, but is most common in old people. Men are affected more commonly than women.

The nature of this neurotic inflammation is still obscure, though many hypotheses have been advanced in regard to it. The fact is that herpes zoster is an acute disease of the nervous system. Modern researches, especially those of *Head* and *Campbell*, make it more and more probable that it is an acute infectious disease with its seat in the spinal ganglia. The morbid agent has a specific affinity for the spinal ganglion analogous to that of the agent of acute poliomyelitis for the substance of the anterior brain. But these things are still wholly unknown. A toxic cause has been ascribed in many cases of herpes zoster, as when associated with carbonic acid poisoning, gout and diabetes. Injuries which have been followed by it have frequently been reported.

The next step in the course of our case will be that the serous contents of the vesicles will become cloudy, then purulent, and finally dry into crusts. This process requires several days, so the period of eruption takes in all about three weeks. As the loss of substance caused by the formation of vesicles extends into the corium scars are usually left, from the characteristic situation of which it can long be recognized that the patient has suffered from an attack of herpes zoster. Herpes zoster gangrænosus is produced in rare cases by the spreading of the ulcers in consequence of secondary infection.

The treatment is essentially symptomatic. We give iodine and arsenic internally to improve the general condition, while we dust the eruption locally with a dry powder and apply boric acid or iodoform ointment. When aspirin and phenacetin are unable to control the severe neuralgic pain we are sometimes obliged to use morphin.

2. Complications on the Part of the Eye in Herpes Zoster

The prognosis of herpes zoster may generally be said to be good, at least when its course is typical, but a number of serious complications may take place in or about the eye which must be taken into account in any particular case.

a. Ocular Complications

An increased secretion of tears ordinarily accompanies the onset. The conjunctiva is commonly injected and herpes vesicles may appear upon it. The prognosis depends mainly on the involvement of the cornea which is affected in about a third of the cases. I have repeatedly mentioned the importance of the trigeminus to the cornea, and after an attack of this disease a paresis of this nerve may remain and a neuroparalytic keratitis may develop still later. Or an eruption of vesicles may appear on the cornea at the same time as the eruption on the skin. It is by no means necessary that the eruption throughout the region supplied by the nasociliary nerve should be simultaneous with the affection of the cornea, according to *Hutchinson*. The cornea alone may suffer, or the herpes may be present along the course of the nasociliary nerve without involving the cornea. In other cases its surface is slightly cloudy, although no defects can be seen in the epithelium. In still other cases the cornea looks as if it had been pricked with needles. Deep, parenchymatous opacities also appear, due to infiltration, the involution of which takes a long time.

It hardly needs to be said that ulcers may develop on a cornea with such lesions through secondary infection. Sometimes all of these complications appear late in the course of the disease, so the cornea needs to be watched carefully throughout, especially as fine opacities may easily escape observation.

The iris may become inflamed either secondarily to the affection of the cornea, or independently when the cornea is not involved. A subacute scleritis may also be met with. Inflammations of the optic nerve resulting in atrophy have been observed, and almost always the intraocular tension is reduced. This hypotony sometimes lasts a long time.

b. Extraocular Comvlications

Chief among these complications are pareses of the nerves of the ocular muscles, the oculomotor, facial and abducens. In old people the disease is sometimes fatal. An important point is that in many cases neuralgias, paræsthesias and anæsthesias persist in the region supplied by the first and second branches of the trigeminus. The sensitiveness of the conjunctiva and cornea may be lost for a long time and the supraorbital neuralgias are extremely obstinate. I know a patient who has suffered many years from such a neuralgia which followed an attack of herpes zoster ophthalmicus.

160. Physiological Importance of the Orbicularis and Spasm of This Muscle

The orbicularis is supplied by the facial nerve and is the muscle that closes the palpebral fissure. Morbid disturbances in its activity consist on the one hand of spasms, on the other of paresis. In order to understand the clinical pictures presented we must first recall to memory some points in the normal action of this muscle.

I. Physiological Importance of the Orbicularis

This muscle is one of the most important protective organs of the eye; when an object approaches it covers the anterior and lateral portions of the eyeball by a closure of the lids. The movement of the lids cleanses the cornea of dust and desquamated epithelium, keeps the moisture of the cornea uniform, prevents it from becoming dry, and also drives the tears toward the inner canthus. When these important functions are impaired the eye must suffer severely.

The orbicularis can also improve the vision by the contraction of the palpebral fissure. We often see this done, for instance by myopes, but the movement cannot be carried too far, for if it is, vision will be interfered with by the irregular diffraction of the light by the cilia.

The closure of the lids in a healthy person is partly voluntary, partly reflex. We can readily convince ourselves, from the intensity with which we can close our eyes, that the action of the orbicularis is complicated. Not only the lids, but the neighboring parts of the skin as well may move, because the orbicularis is connected with the corrugator supercilii, the levator labii superioris alæque nasi, and the zygomaticus minor. When the levator palpebræ is relaxed the upper lid is moved downward by the peritarsal fibres of the orbicularis, and then drawn toward the nasal side by the contraction of the epitarsal fibres. The latter movement alone takes place in the lower lid. The entire orbicularis contracts only when we squeeze our lids together. The margins of the lids are pressed firmly together by the palpebral fibres, while the simultaneous contraction of the orbital fibres causes two folds to appear in the skin, one above, the other below, which are called covering folds, and produces a sort of second closure of the eye, in which the corrugator supercilii takes part, but the frontal portion of the occipitofrontalis does not. The latter muscle opposes the drawing together of the skin over the eye. It is also to be seen in this voluntary closure of the eyelids that the skin is displaced at the outer canthus and that numerous little folds are formed in the skin of the lower lid. If we close our eyes gently, as in sleep, we innervate only the palpebral portion of the orbicularis.

The mechanism of the reflex closure of the lids is constantly in action. The eyes close involuntarily when a foreign body suddenly appears in the visual field, the direction of movement of which is not immediately recognized and may threaten to strike them, or when dazzling light falls upon the eye. This reflex is excited by way of the optic nerve. Under certain circumstances we are able to utilize this reflex for purposes of diagnosis, when it serves to establish a hemianopsia due to disease in the occipital lobe although the patient is in a condition of stupor. The reflex arch between the optic and facial nerves passes through the optic centre of perception in the cortex of the fissura calcarina. There is a sort of reflex closure of the lids excited through the optic nerve which may be observed in many men who sneeze suddenly when brought from the dark into a bright light. A vigorous closure of the lids always follows the sneeze. Photophobic children suffering from keratoconjunctivitis eczematosa are often obliged to sneeze suddenly when we open their eyes. It is also due partly to this that many patients with myopia or astigmatism correspondingly regulate the width of the palpebral fissure. The reflex closure of the lids to moisten and cleanse the cornea is excited through the trigeminus. This reflex is utilized to test the depth of general anæsthesia by touching the conjunctiva and cornea. Slight sensory irritations suffice to excite a reflex action of the orbicularis from the surface of the eye. Under normal conditions an irritation of the extremely minute fibres of the trigeminus in the cornea induces a closure of the lids, as may be seen from the effect produced by a foreign body in the eye. This reflex closure is always common to the lids of both eyes. Many men are unable to voluntarily close one eye alone.

Other muscles make corresponding movements coincidently with the contractions of the orbicularis, a fact which appertains to the physiological action of this muscle. As soon as an attempt is made to close the lids both eyes immediately turn upward and outward; this shows that there is an intimate connection between the innervation of the orbicularis and that of the elevators of the eye, especially of the inferior oblique. This movement places the eye beneath the protecting upper lid when the eyes are closed.

Another movement concomitant with the contraction of the orbicularis is the contraction of the sphincter pupillæ, which has recently been studied by *Westphal.* If, for example, we hold apart the lids of a tabetic patient and direct him to close his eyes strongly a distinct contraction of the pupil can sometimes be observed. Independently of *Westphal, Piltz* has demonstrated this phenomenon in 35% of healthy persons.

II. Spasm of the Orbicularis

This patient is an elderly woman, who comes to us on account of a constant spasm of the lid. Peculiar tonic contractions of the orbicularis take place in both eyes at short intervals, and the eyes are closed for a considerable length of time. The patient states that she is absolutely helpless on the street because of this spasm. Otherwise her eyes are perfectly normal. The condition here is quite similar to that found in an old teacher whose retirement I had to recommend on account of such a blepharospasm. He was nearly run over on the street on one occasion because he was absolutely helpless at the moment of the spasm. If we study the symptoms in this woman a little more carefully we see the margin of the lower lid rise at the moment of the attack, while the upper lid sinks at the same time. The palpebral fissure is drawn to a certain degree to the median side. Curved transverse folds form in the skin of the lid and in the vicinity of the eye. It is interesting to watch the efforts of the patient to relieve the spasm. She first presses up the skin of the forehead with her fingers, for the corrugator supercilii and the frontal portion of the occipitofrontalis are strongly contracted, and in addition she calls upon other muscles supplied by the facial for help by distorting her face and opening her mouth.

Next is a child who presents the well known picture of blepharospasm due to keratoconjunctivitis eczematosa.

It is not without design that I have presented these two cases together. Each represents a type of the two principal varieties of blepharospasm. Spasm of the orbicularis is either a functional disturbance, or it is caused by an organic lesion, so the first question to be answered when we meet with such a condition as this is: Is the trouble functional or organic?

1. Spasm of the Orbicularis as a Functional Disturbance

Many healthy people exhibit a peculiar vibratory tremor of the lids when we tell them to close their eyes gently, as if asleep, and to keep them thus closed. This is known as *Rosenbach's* phenomenon. The corrugator supercilii and the other muscles supplied by the facial nerve are usually involved. The symptom is more distinct in neurasthenia.

Many persons with perfectly healthy eyes frequently have repeated fibrillary twitchings of individual bundles of fibres of the orbicularis which can be distinctly felt. The bundles of fibres may also be seen to shimmer through the skin. Timorous persons are often worried by them, but these fibrillary twitchings are of no importance, they disappear of themselves. Sexual excesses, or hard near work are frequently the causes in persons who are anæmic or neurasthenic. Sometimes we can induce such twitchings artificially by the instillation of physostigmin into the conjunctival sac. If treatment seems to be necessary we may massage the lids, or prescribe small doses of bromide.

There are conditions in which the twitchings of the muscular fibres of the orbicularis are constant, and then we speak of a muscular tremor, or myokymia. This occurs in the course of facial pareses, migraine, etc. The cases in which a true, essential blepharospasm is met with are to be judged quite differently.

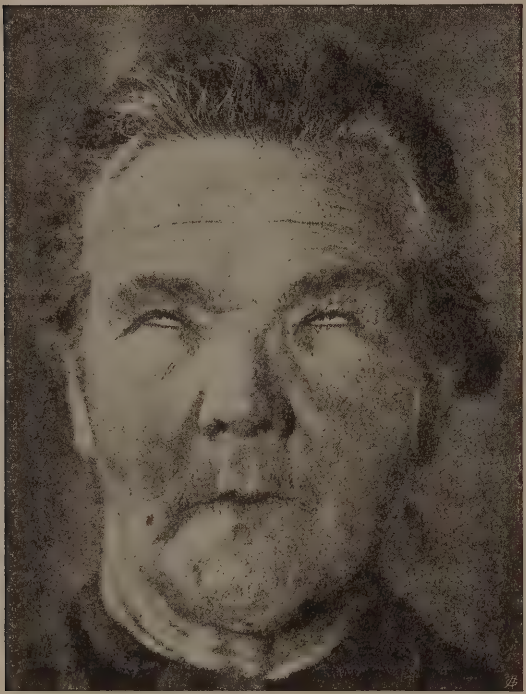


FIG. 129.—Blepharospasmus Senilis at the Moment of the Yielding of the Spasms.

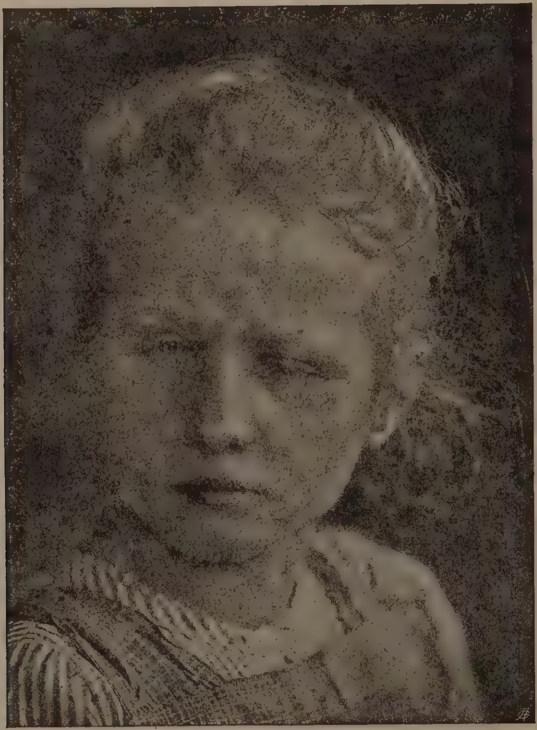


FIG. 130.—Blepharospasmus in a Keratoconjunctivitis Eczematosa of One Eye.

Such a blepharospasm may appear in the form of clonic spasms, which produce a constant blinking, or the spasm may be of a tonic nature, when the eyes are kept closed for a length of time. Clonic spasms of the orbicularis are known as nictitatio, tonic as true blepharospasm. We meet with such functional spasms of the orbicularis in chorea. Here we may also mention the so called habit spasm. Some persons who are thus affected have in addition to the blinking of the eyes, twitchings of the mouth, and shaking movements of the head, a combination that is known as habit chorea. Another kind of habit spasm is to be seen in individuals who habitually try to cover their eyes, with which they see badly, by an incomplete closure of the lids.

Blepharospasm is also observed in migraine and in epileptic convulsions. The tic convulsif of hysteria is very interesting. It may occur only on one side, but is usually present on both. The eyes are closed suddenly and remain so until they open just as suddenly. It has been known for a long time that this hysterical blepharospasm can be arrested by pressure on certain tender points which ordinarily lie along the course of the trigeminus; if pressure is made at the places of exit of the supraorbital or infraorbital nerve the eyes open as if by magic, frequently to close again as soon as the pressure is removed. These points, sensitive to pressure, are often situated elsewhere. I have seen one patient in whom pressure on the tip of the nose stopped the blepharospasm. Such points have been observed in the nose, in the mouth, on the cartilages of the ribs, on the vertebræ, etc. This hysterical blepharospasm must not be mistaken for ptosis. If we lift the upper lid we feel the resistance made by the orbicularis. The eyebrow is situated lower and vertical folds are present in the skin of the forehead, corresponding to the contraction of the orbicularis. In ptosis, on the contrary, the skin of the forehead is cast into horizontal folds by the contraction of the frontal portion of the occipitofrontalis, and the eyebrow is higher. Photopsia, asthenopia and contractions of the extrinsic muscles of the eye frequently accompany a hysterical blepharospasm. Trivial irritations often suffice to relieve a severe spasm of the orbicularis in nervous or hysterical persons, and on the other hand a blepharospasm may be excited in hysterical people by pressure on various parts of the body.

When we consider that under normal conditions the entrance of strong light into the eye causes a reflex closure of the lids it is not surprising that a spasm of the lid may be excited by irritation of the optic nerve in nervous or hysterical persons. Such people are often dazzled by diffuse daylight. Opacities of the refractive media, like those produced by an incipient cataract, may cause symptoms of dazzling and spasms of the lid.

We meet with functional spasm of the orbicularis in the traumatic neuroses, often after very slight injuries to the head. Neurologists have also observed it in spontaneous tetany, in paramyoclonus multiplex, and in myotonia. We sometimes see a sort of occupational spasm of the orbicularis in watchmakers who constantly wear a loupe in the left eye, probably because the abnormal innervation of the facial nerve finally leads to the spasm. Lastly we have blepharospasmus senilis. This must be considered a symptom of age, in so far as other psychogenous causes can be excluded. When we fail to find any organic disease, as in our first case, and the eyes themselves are perfectly normal, we believe this blepharospasm to be functional. As the condition develops in old age we call it senile, and it only remains to be determined whether and in how far psychogenous factors may account for it.

The treatment of functional blepharospasm may be briefly stated as suggestion, arsenic and bromides. In suitable cases, alcohol may be injected at the places of exit of the trigeminal or facial nerves, but I have had no experience with this method.

2. Spasm of the Orbicularis Due to Organic Lesions

Spasm of the orbicularis is due in very many cases to demonstrable organic lesions. It is the most common of all forms of spasm. When we come to investigate in any given case by what organic lesion it is excited we must bear in mind that it may be brought about in two different ways; either by a direct irritation of the facial nerve, or indirectly by reflex action from the trigeminus.

Spasm of the Orbicularis from Direct Organic Lesions along the Course of the Facial Nerve

When the cortical centre of the facial nerve is irritated by such a lesion as a tumor, an abscess of the cerebral cortex, a focus of softening, a deposit of lime on the anterior cerebral convolutions, or a hemorrhagic cyst, the spasm of the orbicularis does not, as a rule, occur alone, but is apt to be associated with spasms and pareses of one side of the body. Blepharospasm due to a cortical lesion is therefore one of the symptoms of *Jacksonian* epilepsy. Twitchings of the muscles of the face may precede the subsequent pareses when the lesion is situated between the cortex and the nucleus in the pons. The lesion may be in the region of the nucleus of the facial nerve. Thus we see facial spasms appear after central hemorrhages in tabes.

The spasms occasioned by direct irritation of the trunk and branches of the facial nerve are of greater importance. Tumors of the petrous portion of the temporal bone, vascular degenerations and meningitic troubles have to be taken into account. Diseases of the middle ear may give rise to clonic twitchings of the orbicularis. Tabes occasionally causes facial spasms. The trunk of the facial nerve is irritated in meningitis, by diseases of the middle ear, and by tumors and aneurysms at the base of the brain. Scars on the forehead and cheeks may cause the greatest variety of spasms of the orbicularis by the pressure and tension they exert on the nerves. For the sake of completeness it may be mentioned that a severe spasm of the muscles of the face may be an important symptom in the course of tetanus.

Reflex Spasm of the Orbicularis from Irritation of the Trigeminus in the Skin, Eye, Mucous Membranes, and Teeth

Any affection of the sensory trigeminus may cause reflex spasm of the orbicularis; injuries, herpes of the skin of the lid, diseases of the nasal cavity, of the teeth, and of the mouth and pharynx may excite it as the result of the irritation produced in the trigeminus. Such a symptomatic blepharospasm accompanies a large number of irritative conditions of the eye. We find it when a foreign body is present in the conjunctival sac, when the cilia scrape the surface of the eye in consequence of trichiasis, and in many forms of inflammation. It is of great practical importance. It is caused in this patient by the fact that numerous fibres of the epithelial plexus of the trigeminus are laid bare in keratoconjunctivitis eczematosa by abrasions of the skin of the lid, and by the detachment of epithelium from the surface of the cornea. This spasm of the orbicularis is associated with photophobia and lacrymation. Often the secretion of the lacrymal gland is greatly increased, so that when the eyes of these children are opened an actual stream of tears gushes forth. The increase of this secretion is to be ascribed to a stimulation of the branch of the facial nerve that supplies the lacrymal gland. Sometimes this blepharospasm lasts for a very long time and the physician should be aware that after the palpebral fissure has been closed in this way for months the children may act as though they were blind, although no pathological lesions can be found in the optic nerve or retina. We must suppose that such children have lost the central attention to optic impressions and that they have to learn again to see. People are met with who once had keratoconjunctivitis eczematosa and relate that when children they were blind at one time.

The presence of such a blepharospasm often makes the examination of the affected eye very difficult, and can moreover exert a bad influence on the entire course of the disease and delay its recovery. This is accounted for by the great pressure that is exerted upon the eyeball. Escape of the conjunctival secretion is rendered difficult, so the eye is cleansed very poorly. Œdema results from the engorgement of the circulation in the lids produced by the compression of the veins passing between the constantly contracted fibres of the orbicularis. Spastic ectropion, or entropion, may likewise develop. It is therefore to be understood that we frequently have occasion primarily to obviate the spasm of the lid. It disappears when we have removed an irritating foreign body, when we have cured an inflammation of the eye, or a trichiasis, but in many cases, particularly of keratoconjunctivitis eczematosa, it is very obstinate. When it is dependent on the formation of rhagades at the outer canthus we must first cure these, in the way already described, with the silver stick and ointment. In other cases the spasm gives way as soon as the child can open his eyes. This end can often be attained by instillations of cocain. I will employ in this case an old measure which occasionally can be used with great benefit. The face is plunged several times in succession into cold water and the eyes open as if by magic. In very severe and obstinate cases, especially when the palpebral fissure is abnormally shortened by blepharophimosis, we must cure the blepharospasm by an operative division of the outer commissure, canthoplasty. Such a spasm of the lid that accompanies some other disease of the eye is called a symptomatic blepharospasm.

The blepharospasm of snow blindness belongs to this class. These cases are characterized by lacrymation, photophobia, severe pain, hyperæmia and chemosis of the conjunctiva and a spasm of the lid. The spasm was ascribed by *Berlin* to this conjunctivitis erythematosa. The action of the chemical rays of light results in a loss of epithelium from the cornea, and so causes an irritation of the trigeminus.

Transition cases from symptomatic to essential blepharospasm are met with. Children are frequently brought to us in whom a constant blinking, or a stronger spasm of the lid, was excited at first by a slight conjunctivitis; the inflammation passed away, but the children continued to blink. In these cases we stand on the hazy confines of the hysterical predisposition. The children are rather anæmic and nervous, as a rule, and treatment with tonics and suggestion soon causes the spasm to disappear.

161. Disturbances of Vision and the Course of the Optic Fibres

Neither of the patients we have to study today have anything externally the matter with their eyes, and the condition of the fundus of each is normal, yet listen to their complaints.

It is probable from his history that our first patient contracted syphilis some years ago. He has felt well ever since until within the last few weeks, when he began to complain of occasional headaches. Two days ago he noticed that he could see nothing to the outer side of each eye.

The second patient, a young student, names his disease himself, he says he has a scintillating scotoma. Scintillations appear before his eyes from time to time, which rapidly increase until finally he can scarcely see anything, while at the same time he suffers from dizziness and from pain confined to one side of the head. The attack passes off after about ten minutes and vision returns. Our duty in such a case as this is to investigate what is the matter and where to look for the cause of the visual disturbance. If we are to do this, if we are to utilize for purposes of diagnosis the visual troubles of which these patients complain and that we can find by our objective tests, and if we are to locate their causes correctly along the course of the optic fibres, we must know not only the various forms of visual disturbance to be met with, but also the course of the optic fibres throughout.

I. Three Possible Sites of a Lesion That Impairs Vision

We must determine in all cases: 1. What conjecture can be drawn from the history as given by the patient concerning the localization of the trouble? 2. By what methods of examination can we obtain the objective signs that indicate its seat? The expert listens to the history and seizes at once upon the right instrument, but we must deal with these questions more fundamentally. The anatomical seat of the lesion may be in one of three places: 1, in the refractive media, in front of the light perceptive layer of the retina; 2, in the percipient layer of the retina and optic nerve; 3, farther up along the course of the optic fibres.

1. Site of the Lesion in Front of the Light Perceptive Layer of the Retina

When patients complain of impaired vision we think primarily of opacities in the refractive media, such as maculæ of the cornea. Opacities of the lens and vitreous cause them to see shadows, which move about when the opacities are in the vitreous, and occasion visual troubles that vary in degree according to their cause and size. Refractive errors also cause much trouble. When the patient complains of bad near vision or of getting tired easily the case is one of asthenopia caused by hypermetropia, presbyopia, astigmatism, paresis or insufficiency of the accommodation. A sudden attack of diplopia is usually binocular and due to a paresis of the ocular muscles, but it may be monocular and caused by anomalies in the refractive media, such as luxation of the lens, or incipient cataract.

To demonstrate objectively the site of the lesion in these forms of trouble we use focal illumination with the loupe and glance into the eye with the ophthalmoscope, by means of which we can see the opacities in the refractive media. They appear gray by focal illumination, black with the ophthalmoscope. Errors of refraction are estimated by the usual methods. When the muscles are at fault we use the tests for muscle imbalance and for paresis. When no such faults as these are present we have to ascertain whether the cause of the trouble is not situated in the retina or optic nerve.

2. Site of the Lesion in the Retina and Optic Nerve

When patients complain of seeing flashes or sparks our attention is attracted to the chorioid and retina, also to glaucoma when they see rainbow colors. Distorted vision is almost always due to inflammation or detachment of the retina. So, too, is the appearance of objects as too small or too large, micropsia, or macropsia. Darkenings before the eyes which vary in intensity point to pressure symptoms in the brain from a tumor cerebri, and to diseases of the retina in nephritis, diabetes and arteriosclerosis. If a cloudy veil seems to be hung suddenly before the eye we think at once of detachment of the retina.

Patients who complain of a marked impairment of vision as soon as it begins to grow dusk are usually suffering from some disease of the retina, such as retinitis pigmentosa, or high myopia. On the other hand the totally color blind have better vision in the twilight, but we must remember that a patient who has a little, sharply defined leucoma which ordinarily covers the pupil may be able to see better in the dusk when the pupil is somewhat dilated.

Most cases of blindness are to be ascribed to diseases of the optic nerve and retina. Embolism or thrombosis of the retinal vessels is a probable cause in a case of sudden blindness of one eye, but the cause may be a fracture of the base of the skull in which the nerve has been crushed. An amaurosis of one eye may be almost acute in a retrobulbar neuritis, or in multiple sclerosis. If blindness of both eyes has come on very gradually it is usually due to tabes, or an atrophy of the optic nerve from some other cause. Sudden blindness of both eyes from occlusion of the vessels is very rare.

When the complaints of the patients are such as to indicate troubles like these we do not use focal illumination, or simply cast the light into the eye with the ophthalmoscope, we use instead two other methods. First of all a careful ophthalmoscopic examination of the fundus, which gives us the desired information in these cases. We use perimetry in addition, because diseases of the retina and optic nerve reveal themselves by scotomata in the field of vision. I need only refer to what it reveals in detachment of the retina, glaucoma and atrophy of the optic nerve. In our first patient this test shows that the outer, temporal half of the field of vision of each eye is wanting. This is a case of temporal hemianopsia. The ophthalmoscopic examination reveals a perfectly normal fundus. The optic nerve and retina appear to be perfectly healthy, so the cause of this visual disturbance must be sought for farther up along the course of the visual tract.

3. Site of the Lesion in the Visual Tract

Lesions that affect the upper portion of the visual tract reveal themselves primarily by disturbances in the field of vision. The ophthalmoscope may or may not be of use in these cases. If the disease is situated in one optic nerve between the retina and the chiasm the trouble is confined to one eye. If it is situated just behind the eyeball so that the central vessels of the optic nerve are implicated, as in shot and stab wounds of the orbit, the fact is readily disclosed ophthalmoscopically, but if it happens to be situated farther upward we have to depend chiefly on our examination of the field of vision. We must first glance over the course of the optic fibres in order that we may understand such a condition of the field as this and locate the lesion correctly.

II. Course of the Optic Fibres

The optic nerve passes from the orbit into the cavity of the skull through the foramen opticum, a small bony canal. Beneath the nerve in the foramen lies the ophthalmic artery. The nerve is surrounded on all sides at this place by bony walls and is therefore easily injured by compression, either by a thickening of the bone, or by crushings and lacerations in fractures. In such a case the ophthalmoscope reveals after a while a descending atrophy on the injured side. The optic nerve is enveloped only by the pial sheath in the optic foramen. From this point the nerve passes within the skull to the chiasm, a distance of scarcely one centimetre.

The Chiasm and the Partial Decussation of the Optic Nerves

The two optic nerves unite in the chiasm, a quadrilateral flat formation with four indentations, an anterior, a posterior, a right and a left, which lies above the sella turcica of the sphenoid bone just in front of the infundibulum. A partial decussation of the fibres of the optic nerve takes place in the chiasm. The nature of this is easily understood from the schematic drawing of the optic tracts (Plate XI), and the importance of this anatomical fact will soon appear. If we imagine a vertical plane to be erected on the visual line of the right eye which passes through the fovea and divides the retina into a right, temporal, and a left, nasal, half we find that the fibres coming from the right half of the retina remain on the right side and form part of the right optic tract. These are the bundles of nerve fibres that do not decussate. On the other hand the fibres that come from the left half of the retina of the right eye pass over to the left side in the chiasm and form part of the left optic tract. These are the decussating bundles. The condition is exactly analogous as regards the two halves of the retina of the left eye. Fibres from both eyes are found in each optic tract. The right optic tract contains the fibres from the right half of the retina of the right eye, which do not decussate, and the decussating fibres from the right half of the retina of the left eye. Thus the right optic tract cares for the right side of both retinæ and therefore, according to the law of projection, of the two left halves of the visual fields. The inverse is true of the left optic tract. An object that lies to the left of the median line transmits the stimulation it excites through the right optic tract, and all that we see on the right side is brought to our consciousness through the stimulation of the left occipital cortex.

This important fact of semidecussation was discovered by *Newton*, but was first accurately described by *Gudden* and is generally accepted today. When opportunity offers to examine the chiasm of a man who has lost an eye in early childhood it is found that the atrophy of the nerve extends to both optic tracts. The bundles that decussate in man amount to about two fifths of the entire number of optic fibres, those that do not decussate to about three fifths. The conditions in animals are often quite different; the decussation is total in the lower vertebrates and first becomes partial in the higher mammals. Still other bundles of fibres are found in the chiasm which have nothing to do with those of the optic nerve, but rather form connections between the two hemispheres (*Gudden's* commissure, *Meynert's* commissure). The optic nerves pass out of the posterior side of the chiasm as the optic tracts, which run backward in a divergent direction, wind about the crura cerebri and reach the corpora geniculata externa at the base of the brain, where each divides into two roots.

Clinical observation, experimental research and pathology have shown us that the fibres of the optic nerve which come from the layer of ganglion cells of the retina enter the corpus geniculatum externum, the pulvinar thalami optici and the anterior corpora quadrigemina, and also find their provisional ends in these three large masses of ganglia. We call these three centres—the corpus geniculatum, the pulvinar and the corpus bigeminum anterius—the primary optic ganglia. The fibres coming from the ganglion layer of the retina end with fine arborescent filaments in these ganglia, but the optic tract by no means stops here. These terminal filaments of the fibres of the optic nerve come in contact again with ganglion cells which send out new axis cylinders toward the centre. The second neuron begins and passes to the posterior third of the internal capsule, where the fibres radiate in a fanlike manner and, in the form of *Gratiolet's* optic radiation, reach the cuneus.

The centre of vision, the so called secondary optic ganglion, or the psycho-optic cortical field, lies in the cuneus, on each side of the brain in the cortex of the occipital lobe. *Munk* has shown that the ganglion cells in the cuneus are arranged just as regularly as the terminal filaments of the fibres in the rods and cones of the retina. Neighboring cortical elements of the one correspond to neighboring elements of the other. This is the place where the stimuli received by the retina are transformed into sensual perceptions. Still this is not all, for the simple change of the stimulus into sensorial perception does not suffice for complete mental understanding.

Numerous fibres extend in various directions from the ganglion cells of the cuneus and join the visual to the other mental centres. These are the association fibres. They unite the centres of sensation with those of thought. Through these connections of the visual centre with the centres of thought changes are produced in the ganglion cells of the cerebral cortex upon which memory depends. We can mentally reproduce an object which we have once learned to know accurately. These are the optic images of memory. According to *Nothnagel* the optic centre of perception lies chiefly in the cortex of the cuneus and of the first occipital convolution. Whether the centres for the senses of space, light and color are separated in the occipital lobe is still undecided. A large part of the cortex of this lobe must at any rate be the depot for the optic pictures of memory. Should these areas be destroyed by softening, apoplexy or tumors, a condition would develop which is known as psychic blindness, a rare condition in which the patients can see quite well under certain circumstances, but can recognize nothing. In order to have this condition produced it is necessary that the occipital lobes of both hemispheres be diseased.

III. The Various Forms of Hemianopsia

We understand by hemianopsia the absence of half of the field of vision of each eye. It may be differentiated according to its nature into *organic* and *reflex*, or according to its external symptoms into *heteronymous* and *homonymous*.

a. Heteronymous Hemianopsia

In this patient the temporal halves of the fields of vision are absent; he has therefore a heteronymous, bitemporal hemianopsia. This is the most common form of heteronymous hemianopsia, and it is evident that the seat of the lesion must be in the chiasm.

If we imagine a sagittal section to be made through the chiasm from before backward all the decussating fibres must be cut across while the others remain intact, and the nasal fibres supply the nasal half of the retina of each eye. If these fibres are destroyed or made incapable of conduction the temporal half of the visual field of each eye must be lost, according to the well known law of projection. This explains bitemporal hemianopsia. When the trouble develops gradually we find at first on testing the visual field a relative scotoma for color on the temporal side of the vertical dividing line. The perception of color is lost first, and finally that of white. If we lay the two visual fields of our patient upon each other it will be seen that the two preserved nasal halves form together an approximately round field of vision which differs from the normal binocular field only in a contraction on the temporal sides to about 60°. Bitemporal hemianopsia usually develops outward from the middle line, and the periphery is at first intact.

The next question is: What morbid processes may destroy the chiasm in the median line, or at the anterior or posterior notch? Bitemporal hemianopsia is met with almost wholly in young or middle aged persons. The causes are:

1. Tumors at the base of the brain, especially of the sphenoid bone. Sarcomata are also found on the sella turcica in the anterior notch of the chiasm.

2. Tumors of the hypophysis, to which we may be directed when acromegaly is present at the same time.

3. It has been shown by *Oppenheim* that syphilis of the base of the brain, in the form of a gumma, or of an endoarteritis syphilitica, is one of the most important and common causes. Such a specific process must be suspected when the clinical symptoms fluctuate back and forth and recur periodically. The ophthalmoscopic condition may be negative, or the fundamental disease may have caused a papillitis, or an incomplete atrophy of the papilla.

The binasal form of heteronymous hemianopsia is very rare in comparison with the bitemporal. The chiasm must be attacked on each of its outer sides by two symmetrically placed lesions. It is met with occasionally when a tumor grows about the chiasm and so injures the fibres on each side that do not decussate. As these fibres supply the outer half of both retinæ, such a lesion must produce a binasal hemianopsia, but practically such a condition is very rarely observed. It must be borne in mind that quite symmetrical contractions of the fields of vision take place in tabes, but in spite of the resemblance these should never be mistaken for a hemianopsia.

b. Homonymous Hemianopsia

In homonymous hemianopsia, either the right or the left half of the field of vision of each eye is lost. Such a condition must be caused by a lesion of the optic tract between the chiasm and the occipital cortex. If, for example, the entire left optic tract is destroyed the fibres are annihilated which supply the temporal half of the retina of the left eye and the nasal half of the retina of the right. Nothing to the right of the median line of the two eyes can be seen, and we have a homonymous hemianopsia of the right side. This is called a tractus hemianopsia. We find in such cases that a vertical line passing through the fixation point of each eye divides the visual field into a seeing and a blind half. The hemianopsia is then complete. Two things are worthy of our notice. First, the lesions are not usually situated within the tract, but begin by affecting the periphery from outside. The nerve fibres which supply the peripheral portions of the retina also lie in the periphery of the optic tract. This explains why symmetrical defects in the visual field usually start in the periphery. The other point is that the various morbid processes, such as hemorrhages, softenings and tumors, do not always destroy the entire tract, so that under certain circumstances only symmetrical quadrants are lost from the fields (incomplete hemianopsia). It must be noticed that the apices of such sectors always lie in the point of fixation, for this is the characteristic feature of the form of hemianopsia due to a lesion of the tract, that the lines of division pass through the point of fixation when the hemianopsia is complete and that the apices of the sectors lie in this point when it is incomplete.

The cause of a homonymous hemianopsia may be situated still farther up, in the visual centre of one occipital cortex. A very striking characteristic produced by a lesion in this situation is that even if the whole centre is destroyed the entire half of the common visual field is not lost. On the contrary we find in the point of fixation a minute portion remaining of the field that is elsewhere destroyed. We call this symptom the "preservation of the macula." This preservation of the macula is particularly distinct when a patient with a cortical homonymous hemianopsia of one side develops a second on the other. I saw such a case of bilateral hemianopsia in Wuerzburg. The man was absolutely helpless, could not go about alone and seemed at first glance to be totally blind, and yet he had a minute central trace of a visual field in which he had, when his visual lines were properly directed, a vision of 6/32. This phenomenon is explained by the so called double supply of the macula of each eye in the cerebral cortex, which is more than probable. We may, for example, suppose that the nasal half of the macula of the right eye possesses a centre not only in the left occipital cortex, but has fibres also in the right cortex. This preservation of the macula is characteristic of a complete cortical and subcortical homonymous hemianopsia. In cases of partial hemianopsia in which only small portions of the visual centre are injured, islandlike homonymous defects appear in the fields of vision where they are situated symmetrically, or we find sectors the apices of which do not quite involve the point of fixation. The conditions are not always as simple in practice as in theory.

The hemianopic immobility of the pupils found by *Wernicke* has been used to differentiate the cortical from the tractus hemianopsia. After the right optic tract has been destroyed the direct and consensual reactions of the pupil to light are abolished when light is thrown on the right half of the retina, and are present when it is thrown on the left half, but as we know from the researches of *Hess* that the reaction to light is excited only from a small area about the macula, the question in regard to the practical application at least of the hemianopic reaction must be left open.

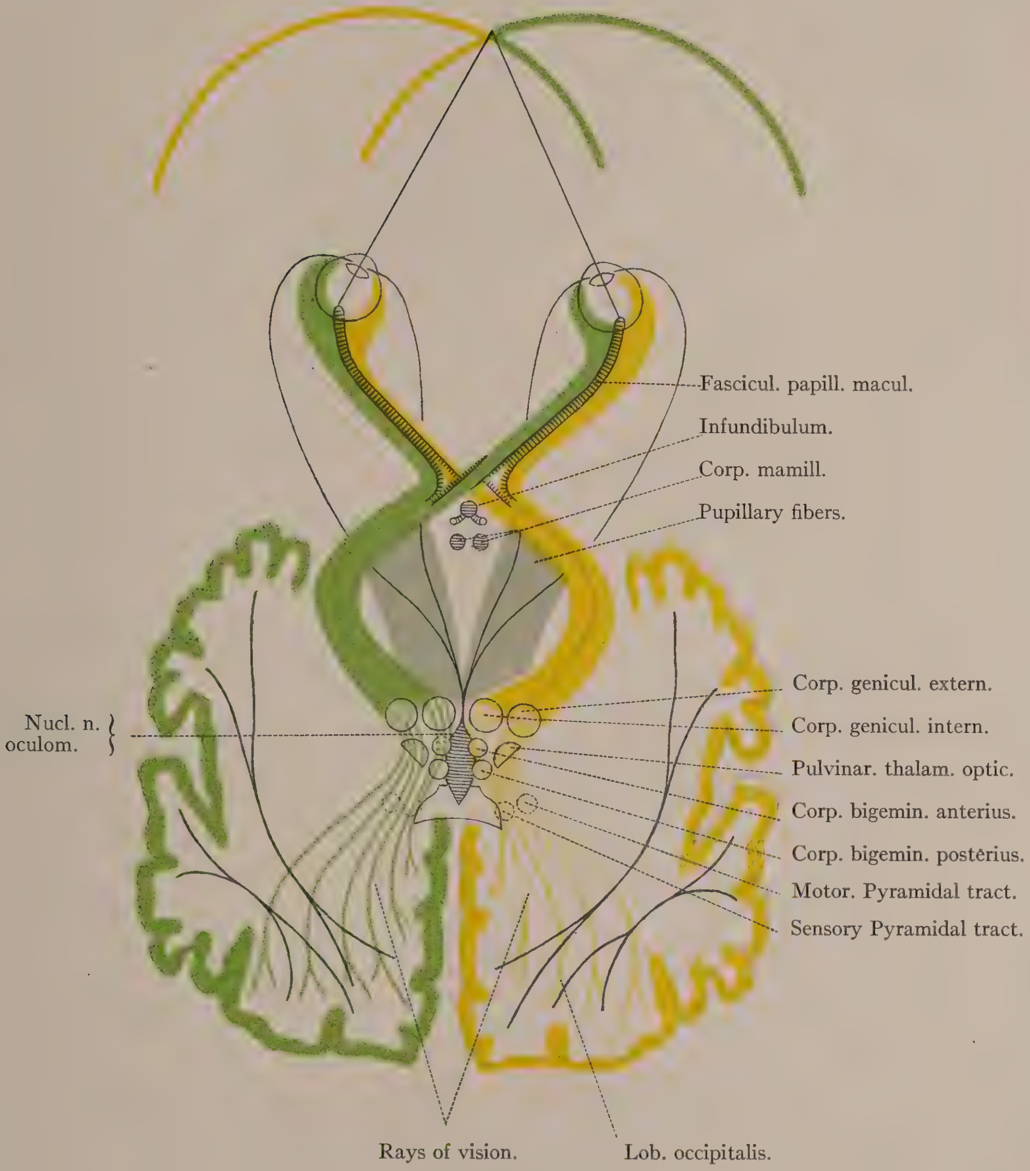
Hallucinatory sensations of sight occur not infrequently on the blind side in cerebral hemianopsia. These indicate that the hemianopsia is subcortical and that the appertaining cortex is still intact, as it can still give rise to hallucinations. A few other practical points are these: If a hemianopsia for color appears in the right side of the field in a case of complete left hemianopsia, the situation of the lesion is indicated to be close behind the chiasm, so that it can involve fibres of the opposite tract. If, in a homonymous hemianopsia, the field of vision of one eye only is gradually lost the lesion is directly in front of the chiasm. If we find disturbances of orientation and aphasia with the hemianopsia the lesion is intracerebral. If it is associated with hemianæsthesia or hemiplegia we locate the disease in the posterior crus of the internal capsule.

Homonymous hemianopsia may have many causes. It sometimes comes on with symptoms of an apoplexy, dizziness, stupor, and headache. Speech may be interfered with when the lesion is at the place indicated by *Naunyn*, where the angular gyrus passes over into the occipital lobe. Such a case belongs to the optic form of aphasia—aphasia with word-blindness. The patient can no longer read even what he himself has written, or he will designate objects by wrong names (paralexia). Motor aphasia may be caused by embolism of the arteria fossæ *Sylvii*. When the hemianopsia is due to hemorrhage improvement may follow the resorption of the blood under certain circumstances, but it does not happen very often. It can be readily understood that reading and writing is considerably more interfered with in a hemianopsia of the right side than in one of the left. In a tractus hemianopsia we think primarily of a tumor of the base of the skull, or of the temporal lobe, in which case pareses of other cranial nerves will be present.

Scintillating Scotoma, a Reflex Hemianopsia

Scintillating scotoma is an angioneurosis, is unmistakably connected with migraine, and for that reason has often been called ophthalmic migraine. The visual disturbance is a sort of hemianopsia. This patient states that at the beginning of the attack a blind spot appears close to the point upon which he is fixing, and that from this place the loss of the field of vision extends toward the periphery, while jagged lines flicker here and there in the area first affected. It is always only one certain half of the field of both eyes that is involved, so this is a sort of hemianopsia, though from a cause entirely different from that which operated in our first case. As soon as the limit of the visual field in the outer half of both eyes has been reached the scintillations cease and vision slowly returns from the centre. In other cases the hemianopsia, in scintillating scotoma, may be homonymous with a vertical line of demarcation. The upper or lower half of the field is also occasionally affected. Again in other patients the hemianopsia is less marked and there is only a uniform, scintillating cloud before the eyes.

The fact that both eyes are uniformly affected proves that the origin of scintillating scotoma must be central. This follows also from the sort of visual disturbance and from the factors that accompany it. The attack lasts on an average from a quarter to half an hour, and is then followed by hemicrania, nausea, and it may be vomiting—symptoms of migraine. It is generally supposed to be due to a trouble of the innervation of the vessels which causes a transient circulatory disturbance, probably in the optic cortical field of the occipital lobe. The irritation of the optic elements produces the scintillations at the same time that the perception of the peripheral stimuli is abolished. In grave cases of this angioneurotic trouble, which is chiefly an anæmia, the disturbance spreads to other central areas and we meet with paræsthesias and impairments of the speech, memory and hearing. According to *Jolly*, who himself suffered from scintillating scotoma, the most common form is brought about centrally from the chiasm, perhaps in the optic tract, the external geniculate body, or the optic radiations. A scintillating scotoma of only one eye is also met with. This must be ascribed to the retina and optic nerve. We can explain the symptoms best as due to transient disturbances in the circulation. Anyone who has ever fainted knows that such circulatory disturbances in the brain excite quite analogous symptoms—green and blue colors and scintillations before the eyes.



Neural routes of vision.

bman Company, New York.

As long as the attacks occur at long intervals they have no serious importance. They are accustomed to become less frequent as age advances. Treatment must be directed toward the general condition of the body, toward a tendency to neurasthenia. Too much mental work and overstraining of the eyes must be avoided. It may be possible to arrest the attack in its beginning by a drink of coffee, tea or wine. Certain drugs are sometimes of benefit, such as quinine, aspirin and antifebrin.

To sum up what has been said concerning the localization of the lesion along the intracerebral course of the optic nerve fibres: The lesion must be situated *in front of the chiasm*, in the optic nerve itself, in all cases in which the loss of the visual field affects only one eye, or when the defects in the fields of the two eyes are not of a homonymous nature. The lesion is *in the chiasm* in temporal hemianopsia, in which the decussating fibres alone are affected. It is *above the chiasm* when the hemianopsia is homonymous, affecting the two right, or the two left halves of the fields of vision.

162. In How Far Can the Ocular Symptoms Contribute to the Localization of a Tumor in the Brain?

This patient complains of a dull, boring headache, of a feeling of dizziness and of vomiting. He has a distinctly slow pulse, signs of stupor and of other psychical troubles, and we find with the ophthalmoscope a marked choked disc in each eye. The diagnosis of tumor cerebri is based on these symptoms. Choked disc is caused in 90% of all cases of tumor of the brain and forms the most common general symptom of this condition. But the general diagnosis of a tumor does not suffice in such a case, we need to determine what sort of a neoplasm it may be, and whether, after exclusion of a syphilitic growth, for which specific treatment is necessary, the patient may perhaps be saved by means of an operation.

It is only in rare cases that a tumor of the brain can be accurately located by means of the ocular symptoms alone, because the conditions are too complicated and the accurate localization of such a tumor is very difficult, as is proved by the numerous mishaps met with in brain surgery. Because of this difficulty I try to make the study of how far the ocular symptoms are at least of use in making this diagnosis by the answers to the following questions: 1. Does choked disc furnish any points of value in the diagnosis of the site of a tumor? 2. May a tumor be situated in such a part of the brain that ocular symptoms are not produced? 3. May it be in a portion of the brain where the ocular symptoms produced are at least worthy of attention? 4. May it occupy a region in which the ocular symptoms have a determining value?

I. Does Choked Disc Furnish Any Diagnostic Points in Regard to the Site of the Tumor?

This question is naturally the first to be taken into account because choked disc is not only the most important general symptom, but also the commonest ocular symptom of a tumor of the brain. Visual troubles appear early in the choked disc of tumors only under two conditions, in the absence of which the vision may remain intact for a long time in spite of the greatly swollen state of the head of the optic nerve. These conditions are: 1, When the tumor is situated in the cerebellum, or in the angle between the cerebellum and the pons; 2, when it is at the base of the brain and involves the optic tract directly. The reasons for this are that the symptoms of intracranial pressure reach a very high degree quite early in the course of tumors of the cerebellum and of its vicinity, and it is self evident that the signs of engorgement in the papilla may be very marked in direct lesions of the optic tracts. Hence if we find among the general symptoms that indicate a tumor of the brain a very early high degree of choked disc, possibly with hemorrhages, white patches of degeneration in the retina and serious visual trouble, the situation of the tumor is suggested as being either in the cerebellum, in the angle between the cerebellum and the pons, or at the base of the brain in the region of the chiasm and of the cavernous sinus.

Although it is the most important and most common ocular symptom of a tumor of the brain, choked disc does not otherwise appear early. In tumors of the cerebrum, especially of the frontal portion, of the corpus callosum, of the central convolutions, of the temporal convolutions, and, according to *Oppenheim*, of the occipital portion, no early symptom is usually present. We can therefore draw no conclusion as to the size of the tumor from the degree of engorgement in the disc; very large tumors may exist without producing any sign of a choked disc, while a very small growth may cause a great engorgement. Hence the presence of a choked disc is in general only confirmatory of the general diagnosis of a tumor of the brain.

We are not much better off when in the course of a tumor of the brain we try to utilize for diagnostic purposes either a transitory or a more or less complete attack of blindness. Such a visual disturbance may be caused by a great engorgement in both optic nerves, as for example, when the floor of the third ventricle is arched forward so as to press directly on the chiasm. But a lesion in the occipital lobe may also cause a temporary disturbance of vision. I have seen blindness appear periodically in a case of sarcoma of the occipital lobe. Homonymous hemianopic contractions of the field of vision, sometimes in the form of scintillating scotoma, may indicate that the tumor is situated in this region.

In addition to the choked disc we find in many cases other important ocular symptoms, such as disturbances in the movements of the eyes, pareses and anomalies in the visual field.

II. Portions of the Brain in Which a Tumor Causes No Ocular Symptoms

Tumors that excite no ocular symptoms of diagnostic importance, aside from a choked disc, are found, 1, in the central convolutions; 2, in the frontal portion of the brain; 3, in the region of speech; 4, in the corpus callosum; 5, in the ventricle.

Tumors of the Central Convolutions

The symptoms produced by tumors of the central convolutions are essentially those of *Jacksonian* epilepsy. The spasms are confined at first to the centres affected, then spread to other centres, according to the localization of the tumor in the cerebral cortex, and are followed by monoplegias in the muscular regions affected, and then by hemiplegia.

Tumors of the Right Temporal Lobe

Ocular symptoms may be said to be absent in these cases, for it seems to be doubtful whether the connection, to which attention was called by *Knapp*, of partial paresis of the oculomotor nerve on the same side as the lesion, hemiplegia of the opposite side and cerebellar ataxia is diagnostic.

Tumors of the Left Temporal Lobe and of the Region of Speech

The same is true of tumors of the left temporal lobe and of the region of speech, which includes the left third frontal convolution, the island of *Reil*, the posterior end of the first temporal convolution, parts of the angular gyrus and many subcortical association tracts. The disturbance of speech, especially sensory aphasia, is the most important focal symptom.

Tumors of the Corpus Callosum

These tumors present so few characteristic symptoms that they are rarely diagnosed. Bilateral pareses of the limbs, without involvement of the cerebral nerves and very serious impairment of the intelligence, are the most characteristic. They cause no ocular trouble.

Tumors of the Cerebral Ventricle

The tumor most frequently met with in the ventricle is the one produced by the cysticercus. The symptoms are so uncertain that a diagnosis can very rarely be made. When the lesion encroaches on the cerebellum or the pons the symptoms thus produced predominate.

III. Portions of the Brain in Which a Tumor Causes Ocular Symptoms That at Least Deserve Notice

Ocular symptoms, although not of conspicuous importance, are produced by tumors in the centrum semiovale and the cerebral ganglia in the frontal portion of the brain, in the parietal lobes and in the angle between the cerebellum and the pons. When a tumor in one hemisphere has involved the corpus caudatum, the corpus lenticulare and the thalamus opticus, a hemianopsia, a mydriasis, or some other partial oculomotor paresis of the opposite side may be present in addition to the hemiplegia of the opposite side, the unilateral tremor and the unilateral facial paresis. In such a case the hemianopsia is to be explained by a lesion of the corpus geniculatum externum, or of the optic radiations. The pareses of the ocular muscles in tumors of the thalamus are to be ascribed to lesions of the nuclei of the oculomotor nerve, or of its trunk at the base of the brain.

Tumors in the Parietal Lobe

Isolated pareses of the oculomotor nerve, either on the same or on the opposite side, occur in connection with tumors of the parietal lobes, but they are usually to be considered as distant effects produced by compression either of the nerves at the base of the brain, or of the corpora quadrigemina.

Tumors in the Frontal Portion of the Brain

It is only when a tumor of the frontal portion of the brain grows toward, or develops from the base that it can exert pressure upon the optic nerves, the chiasm and the optic tract, and so produce a choked disc in one eye alone, crossed or bitemporal hemianopsia, or neuralgia of the first branch of the trigeminus. Choked disc is usually a late symptom of tumors in this portion of the brain.

Tumors of the Auditory Nerve in the Angle between the Cerebellum and the Pons

Comparatively little can be contributed to the accurate localization of these tumors by the ocular conditions. Double choked disc is present, with or without signs of neuritic atrophy, in the overwhelming majority of cases, in as much as 90%, but it is just as frequent with tumors of the posterior fossa of the skull, so that only its relatively early appearance can be of any diagnostic value. Pareses of the ocular muscles are of much more rare occurrence, because these tumors are encapsuled and do not encroach upon the centrally placed optic tract. Of these muscles the abducens is rather the more apt to be affected in either one or both eyes. Paresis of the associated movements of the eye laterally seldom occurs. The facial is the nerve most often involved, as would naturally be expected from its close proximity to the auditory, but, in contrast with the condition found in diseases of the pons this facial paresis almost never occurs in connection with paresis of the body on the opposite side. The involvement of the trigeminus is insignificant, and *Ziehen* has shown that trigeminal pain is absent with tumors of the auditory nerve, a condition which contrasts sharply with that produced by tumors which spring from the place of exit of the trigeminus itself. In tumors of the auditory nerve it is the impairment of hearing that appears early, while the pareses of the ocular muscles take a subordinate position. A central deafness, a simultaneous paresis of the auditory and facial nerves, perhaps with involvement of the trigeminus, but above all with involvement of the cerebellum, as shown by disturbances of equilibrium and ataxias, call attention to this form of tumor in the posterior fossa of the skull.

IV. Portions of the Brain in Which a Tumor Causes Ocular Symptoms That Have a Diagnostic Value

Tumors of the Crus Cerebri

The most frequent lesion met with in this locality is the solitary tubercle of childhood, less often softenings and hemorrhages are to be observed. The most important symptom of disease of the crus cerebri is oculomotor paresis on one side and paresis of the opposite side of the body. This is *Weber's* symptom complex. The oculomotor paresis may appear first, and then the hemiplegia, or the order may be reversed, but, as a rule, the two occur simultaneously. If the lesion is in the lemniscus the oculomotor paresis may be associated with ataxia, without paresis of the limbs on the opposite side of the body, as found by *Bruns*. All the branches of the oculomotor nerve are usually affected, but occasionally it happens that the branches to the internal muscles of the eye are spared. As the portions of the nucleus for the sphincter and the muscle of accommodation are situated the farthest forward, the fibres that supply these muscles must take a special course. If several foci are present in the peduncles, or if the lesion involves both crura, the oculomotor paresis may be bilateral, but such cases are rare. Another point of importance is that ptosis may be the first sign of the affection of the oculomotor nerve in a commencing disease of the crus. This syndrome of *Weber's* prevails so in lesions of the crus cerebri that a hemiplegia without oculomotor paresis, or inversely oculomotor paresis without hemiplegia, scarcely needs to be taken into account, although such cases have occasionally been observed. Nystagmus is almost never present, and the ophthalmoscope reveals nothing decisive. Neuritis has been observed in only about 10% of the cases, and at all events disturbances on the part of the optic nerve are more rare with diseases of the crus cerebri than with affections of the pons. *Marburg* sums up the subject in the following words: "*Weber's* syndrome, in its complete development (contralateral hemiplegia with involvement of the cerebral nerves and with total oculomotor paresis on the same side as the lesion), indicates a disease of the median portion of the peduncle. When the oculomotor paresis is incomplete (the pupil remaining unaffected) the lesion is confined to the spinal portion. If disturbances of sensibility are added the lesion extends into the tegmentum, while with the vasomotor trophic disturbances, to which a single interpretation cannot be given, the middle segments of the peduncle motor area, or the closely adjacent substantia nigra must be affected." Tumors of the crus cerebri often extend to the other side, and we have then a bilateral oculomotor paresis with hemiplegia, and later, signs of paraplegia as the other crus becomes involved.

Tumors of the Pons

A tumor of the pons is diagnosed from the presence of an alternating hemiplegia, by which is meant paresis of one or more cerebral nerves that leave the pons on the side of the tumor together with paresis of the limbs and of the nerves that leave below the pons, particularly the hypoglossus, on the opposite side. The form of paresis is rendered possible by the fact that in the region of the nuclei of the nerves in the pons the pyramidal tract from which the paresis of the limbs proceeds is still on the same side.

The most common form of alternating hemiplegia is *Gubler's*, in which the facial nerve is paretic on the side of the tumor, while the limbs and the hypoglossus on the opposite side are affected. Another form is one in which the abducens on the side of the tumor and the limbs of the opposite side are paretic. The tumor must be situated in such a case at the place in the pyramidal tract through which the roots of the abducens pass. A third form is a lesion of the trigeminus on the side of the tumor and hemiplegia of the opposite side. We can also diagnose a tumor of one side of the pons from an inability of the two eyes to turn past the middle line toward the side on which the tumor is located. In such a case the tumor is in the region of the nucleus of the abducens. Sometimes only the associated lateral movement of the eyes is paretic, but the power of convergence of the affected internus may also be compromised. This paresis of the associated lateral movement of the eyes may be present alone, or it may be combined with paresis of the limbs on the opposite side. Still other combinations of these ocular symptoms may be met with in tumors of one side of the pons. Thus a paresis of the facial nerve and of the abducens, or a facial paresis with paresis of the associated lateral movement of the eyes, may be combined with paresis of the limbs on the opposite side. When the tumor extends beyond the middle line of the pons the symptoms become more complicated. Then we may have paresis of the lateral associated movement of the eyes in one direction added to that in the other, so that the eyes remain immobile in the middle line, or we may have hemiplegia of one side added to hemiplegia of the other. The symptoms are particularly complex when we have to deal with diffusely growing gliomata.

Tumors of the Corpora Quadrigemina and of the Glandula Pinealis

The ocular symptoms excited by tumors of the corpora quadrigemina and of the pineal gland are of conspicuous importance. *Nothnagel* taught us long ago that pareses of the ocular muscles, an impairment of equilibrium in walking or standing, and a deafness on one side are the most important symptoms diagnostic of the localization of tumors in the corpora quadrigemina. A tumor in the region of the corpora quadrigemina and pineal gland causes a hydrocephalus internus and thereby a choked disc, by the compression of the aquæductus *Sylvii* and of the vena *Galeni.* A disturbance of the ocular muscles, especially of those supplied by the oculomotor nerve, is met with, but the most characteristic symptom is a paresis of the associated movements of the eyes upward and downward, which has been observed in almost 20% of the cases. Paresis of the associated lateral movements of the eyes almost never occurs, contrasting sharply with the fact that it forms such a prominent symptom in diseases of the pons. The conclusion has been drawn from this that in man the corpora quadrigemina have no control over the lateral movements of the eyes, but have over the vertical. Ptosis with bilateral paresis of the oculomotor nerve is another very important symptom of disease in the corpora quadrigemina, while the trochlearis and abducens are rarely affected. Disturbances of the pupils and of their reactions are very frequent, and are due to the lesions of the oculomotor nerve and the visual troubles. It may be said that according to *Bach* a destruction of the superficial portions of the corpora quadrigemina does not cause a reflex immobility of the pupil to light. Nystagmus and disturbances of hearing are notably frequent. The deafness is due to the fact that the posterior corpora quadrigemina are connected with the auditory nerve of the opposite side through the lateral lemniscus, the upper olive and the opposite corpus trapezoides. It very rarely happens that the trigeminus is affected. A tumor of the corpora quadrigemina is to be suspected whenever an ophthalmoplegia is the most prominent feature of the clinical picture, especially when the muscles of both eyes are involved, associated with a disturbance of equilibrium in standing or walking, and a deafness of one ear.

Tumors of the Cerebellum

Tumors of the cerebellum betray themselves very often by quite positive focal symptoms, the most important of which is the cerebellar ataxia, which appears early and is well marked. The symptom next in importance is the dizziness, which is a true rotatory vertigo. The patients feel as if the objects in their vicinity were rotating about them, or as though they themselves were rotating in space. The next focal symptom is the atactic movements of the limbs. Then come the ocular symptoms, such as nystagmic twitchings, divergence of the optic axes, and pareses of the associated movements of the eyes. The nystagmic twitchings may take place only toward the side of the disease, or may be irregular. Pareses of the associated movements of the eyes toward the side on which the lesion is situated have been observed. In many cases the pareses of the ocular muscles are due to the pressure of the cerebellar tumor upon the nuclear region of the ocular muscles in the corpora quadrigemina, when they have the character of nuclear pareses, a number of the muscles of each eye being affected, although not symmetrically. Paresis of the abducens is frequently absent and the pupil and accommodation may be spared. *Bruns* has met with these nuclear ophthalmoplegias so often in tumors of the cerebellum that he considers it an almost constant symptom.

Tumors of the cerebellum can naturally produce symptoms referable to the neighboring parts, as, for example, to the pons. Then pareses of the associated movements of the eyes appear; the eyes cannot be turned to the left if the lesion is on the left side, the patient cannot look to the right if that is the side on which the tumor is located, while the power of convergence may be retained. Symptoms may be excited on the part of the nerves at the base of the brain when the tumor grows in this direction. These lesions may be distinguished from those produced in the nuclei by the fact that the symptoms are usually confined to one side, and their diagnostic value is due to the fact that they are ordinarily present on the same side with the tumor. The abducens is the nerve most apt to be affected at the base of the brain by a lateral tumor of the cerebellum, while the pareses of the oculomotor are usually due to lesions of the nuclear regions in the corpora quadrigemina.

Oppenheim first called attention to an isolated reflex anæsthesia of the cornea which may be present for a long time as the first sign of a commencing anæsthesia of the trigeminus in connection with tumors of the cerebellum. When it is taken into consideration that the symptom most frequently produced by these tumors is a choked disc, caused by the rapid development of a hydrocephalus internus due to an occlusion of the aquæductus *Sylvii*, it is evident that we must question in every case whether the tumor is not situated in the cerebellum.

Tumors of the Basis Cranii

The ocular symptoms produced by tumors at the base of the skull are of still greater importance. These tumors may be divided into those of the anterior, middle and posterior fossæ. Tumors of the anterior fossa are very rare, but they have one point of interest in that such a tumor may break through the upper wall and penetrate into the posterior part of the orbit, where it will produce the same symptoms as a primary tumor of the orbit; blindness of one eye, exophthalmos, pareses of the ocular muscles, and supraorbital neuralgia.

Tumors of the posterior fossa cause complicated ocular symptoms inasmuch as they may originate from the cerebellum, the caudex cerebri or the nerves at the base of the brain. This is the reason why the point of origin frequently cannot be determined clinically. It is only when the tumor is situated in the angle between the cerebellum and the pons that the symptoms produced are to some degree characteristic, sufficiently so that tumors have repeatedly been removed successfully from this region.

Tumors of the middle fossa produce ocular symptoms that are very much more characteristic because such important structures as the optic nerves, the chiasm, the optic tracts, the nerves to the ocular muscles, and the trigeminus, lie in this region. Tumors of the sella turcica which come from the periosteum, the chiasm and the sphenoidal sinus, cause symptoms which are so characteristic that they give the diagnosis at once. When they are situated in the median line they cause bitemporal hemianopsia in consequence of the lesion they produce in the decussating fibres of the optic nerve. Gummata may cause this hemianopsia to appear suddenly and then disappear. If the tumor is situated farther forward on one optic nerve one eye is rendered blind; if it encroaches backward on the chiasm it causes blindness of one eye and temporal hemianopsia of the other. If the tumor involves an optic tract behind the chiasm homonymous hemianopsia on the opposite side is produced. As the tumors of the sella turcica become larger so as to encroach upon the lateral portions of the middle fossa pareses of the muscles of the eye and disturbances on the part of the trigeminus, such as neuroparalytic keratitis, are added. The first sign of the involvement of the oculomotor nerve often is a ptosis.

Even when the symptoms on the part of the muscles of the eye and of the trigeminus are at first only on one side it almost always happens that the affections become bilateral. Choked disc may be absent throughout the entire course of these tumors, but the hemianopsia, without any lesion to be seen with the ophthalmoscope, followed later by an atrophy of the optic nerve, furnishes sufficient ocular data for the diagnosis of this kind of a tumor.

Tumors of the hypophysis produce at first the same symptoms as other tumors in the sella turcica. When they are situated exactly in the middle they give rise to a bitemporal hemianopsia, but many of them cause a quite specific symptom, the acromegaly described by *Marie*, in which the hands and feet become abnormally large, as well as portions of the face, such as the chin, nose and lips. This acromegaly occurs in simple hyperplasia of the hypophysis, which causes a hypersecretion. Such an acromegaly, associated with bitemporal hemianopsia, locates the tumor beyond a doubt. When the tumors come not from the middle, but from the lateral parts of the middle fossa, the succession of the symptoms produced is different, yet the disease of this structure is present even then.

When we thus investigate in what group of tumor symptoms the ocular symptoms in any case may fall, we see that we can contribute very much to the accurate localization of a tumor in the brain.

163. The Ocular Symptoms Which Indicate That a Disease Is Situated in the Pons Varolii

The ocular symptoms in this boy, 14 years old, were not only the first signs of disease, but they indicate positively a certain lesion when taken in connection with other symptoms. The boy has hitherto been perfectly well, but he comes of a tuberculous family. His trouble began with a paresis of the left abducens, the cause of which was at first uncertain. Some four or five weeks later a paresis began and has steadily increased in the limbs on his right side. Then he began to have attacks of dizziness and sometimes vomited, especially in the morning. Nothing pathological can be found with the ophthalmoscope. With this combination, paresis of the abducens on one side, paresis of the limbs on the other, the ophthalmologist must suspect the presence of a lesion in the pons. This diagnosis has been confirmed in the neurological clinic, the report of which reads: Well built lad, in a fair state of nutrition. The right leg is held stiffly in walking, the outer edge of the foot touches the floor first, the foot is carried forward in a semicircle. Walk not made worse by closing of the eyes. Stands firmly on the left foot, wavers a little on the right. *Romberg* indicated. Maximal circumference of right leg, 26 cm; of left, 26½ cm; circumference of right thigh, 16 cm above the patella, 36 cm; of left, 36 cm. During rest the right foot assumes a position of slight varoequinus. Paresis of the tibialis anticus, of the peronei, and of the muscles of the calf. The toes can move only a very little. Muscles of the thigh in good condition. No ataxia, changes of position promptly perceived. Clonus of right patellar reflex from slight blows. Clonus of the reflex of the tendo Achillis on both sides. Stroking of the sole of the left foot produces an isolated contraction of the tensor fasciæ latæ and plantar flexion of the toes; of that of the right foot a strong dorsal flexion of the great toe and feeble extension of the others. No spasms are excited by passive movements. Cremasteric and abdominal reflexes more lively on the left side than on the right. The left shoulder is rather lower than the right. The right arm cannot be raised as high as the left, and tires more quickly. Slight paresis of the right triceps, biceps and of all the muscles of the forearm and hand. Circumference of the middle of the right arm, 19½ cm; of the left, 20 cm; maximal of the right forearm, 19½ cm; of the left, 20 cm. No ataxia; no disturbance of the sensation of posture; no tremor; periosteal and tendon reflexes greater on the right side than on the left. Direct muscular excitability greater on the right side than on the left. All qualities of sensibility good, except that extremely gentle touches of the pencil on the right hand are sometimes not perceived. Movements of the head free and not painful, even when he shakes his head. No sensitiveness to pressure or percussion. Tongue deviates to the right; no atrophy; no tremor. Uvula deviates a little to the left. Palate raised well on intonation. Pharyngeal reflex absent. The left cheek puffs out more than the right. Innervation of the facial muscles good. The right eye closes the more strongly; a narrow slit remains open in the left when the eyes are closed gently as in sleep. Paresis of the left abducens, movements of the eye otherwise good. Reaction of both pupils to light and accommodation equal and good. Corneal reflexes of both eyes weak, that of the right less than that of the left. Tender points of the trigeminus not painful on pressure on either side. Hearing of both ears normal. Taste intact. Left masseteric reflex seems to be the stronger. Muscles of mastication normal. Condition of internal organs negative. No albumin or sugar in the urine. Quantity of urine not increased, no polydipsia.

Two classes of diseases of the pons are met with in practice: 1, tumors; 2, other focal diseases due to hemorrhages, thrombotic softenings and abscesses. We are able to answer the question as to what ocular troubles indicate a lesion in the pons more precisely now than formerly, thanks to the joint labors of neurologists and ophthalmologists. *Uhthoff* in particular has recently sifted, critically, the immense mass of material and has compared the findings at autopsies with the clinical observations made on the living patients, and has thereby done us a great service.

A. Ocular Troubles in Tumors of the Pons

The solitary tubercle, especially of young people, is the most common of all the tumors of the pons; then follow gliosarcoma, sarcoma, and gumma. The cardinal ocular symptoms produced by one of these tumors are:

1. *Choked Disc.*—This is present in more than half of the cases. Optic neuritis is also very common, though less frequent than choked disc. It is of diagnostic interest to know that true choked disc is not as apt to appear in connection with solitary tubercle as with gliosarcoma of the pons.

2. *Paresis of the Abducens with Affections of the Other Cerebral Nerves, Especially of the Facial.*—This combination is present in more than 75% of the cases. The paresis of the abducens may be bilateral, while only one facial nerve is affected.

3. *Paresis of the Opposite Side of the Body.*—The paresis of the abducens may be combined with that of the internal rectus of the other eye, so as to produce a paresis of the lateral associated movement of the eyes. This paresis of the associated lateral movement, taken together with a paresis of the opposite side of the body, must be looked upon as one of the most important diagnostic symptoms of a tumor in the pons. It is present in more than a third of the cases. The reaction of convergence may be perfectly intact; the same internus that is paretic in the lateral associated movement may act in convergence. This shows that the lateral movements and the convergence must have different centres of innervation. The paresis of the associated lateral movements may be bilateral when several tumors are present, or when a tumor has encroached upon the other side of the pons.

As regards the other ocular nerves the oculomotor is affected with comparative infrequency, but when it is involved the paresis of the body is usually on the opposite side. In such a case we may suppose that either a tumor of the pons has encroached upon the pedunculus cerebri, the nuclear region of the oculomotor nerve in the floor of the aquæductus *Sylvii,* and the region of the corpora quadrigemina, or that a neoplasm from these regions has attacked the pons. Sometimes only a ptosis with paresis of the opposite side of the body is present, but the ptosis may be due to other causes. It rarely happens that a bilateral paresis of the movements of the eyes upward and downward is caused by a tumor of the pons. The facial nerve is affected much more often than the oculomotor, as has been said. A combination of paresis of the facial nerve with paresis of the opposite side of the body (*Millard-Gubler's* paresis) excites suspicion that a tumor is situated in the pons. Facial paresis, with inability to turn the eyes toward the same side, is also a very characteristic symptom, but if we meet with a combination of pareses of the facial and abducens we are not justified in suspecting a tumor of the pons until after pareses of other nerves on the same side have been added, especially of the trigeminus. The latter nerve is never affected alone in a tumor of the pons, but always in combination with other cerebral nerves. When both it and the facial are involved by a tumor in this situation a neuroparalytic keratitis may develop, but this combination is not necessarily indicative of such a tumor. The pupil furnishes no signs of the presence of a tumor in the pons, and nystagmus has only occasionally been observed.

B. Ocular Troubles in Other Focal Diseases of the Pons

The clinical pictures produced by hemorrhages, thrombotic softenings and abscesses in the pons can be differentiated from those caused by tumors by the fact that the onset of the symptoms is more acute, is frequently sudden, and that they reach their maximum more quickly. Those focal diseases are less common than tumors. *Uhthoff* found 178 tumors of the pons revealed by autopsy to 51 cases of other focal diseases. Otherwise the symptoms are the same. The inability to turn the eyes laterally, combined with paresis of the opposite side of the body and with involvement of the facial and trigeminus of the same side, remains characteristic. A striking fact is that a neuroparalytic keratitis is met with very rarely in these diseases, in spite of the pareses of the facial and trigeminal nerves. The pupils afford little help, but meiosis seems to be frequent. Reflex immobility of the pupil to light, with preservation of the reaction to convergence, must be said to be very rare. The changes in the optic nerve and fundus aid us just as little, except that a neuritis is frequently observed in connection with an abscess of the pons, but these abscesses again are very uncommon. A true choked disc, or an atrophy of the optic nerve, together with symptoms referable to the pons, is indicative rather of a tumor.

164. In How Far Can Ocular Symptoms Be Utilized in the Diagnosis of a Meningitis?

The vision of this patient is greatly impaired by an optic atrophy. He states that more than ten years ago he was sick for a long time, suffered from cerebral symptoms, headache, vomiting, dizziness and stupor, and that during the course of this disease he lost the sight of both eyes almost completely. He is now perfectly well. *Wassermann's* reaction is negative. We find in him simply an atrophy of the optic nerve that must be ascribed to a former so called chronic meningitis simplex, from which recovery may take place. In this form of meningitis we depend chiefly for our diagnosis on the visual disturbances and on the changes in the fundus, as these may afford the first explanation of the uncertain cerebral symptoms from which the patients have been suffering. It is not to be denied that in many of these cases the changes in the optic nerve can be ascribed to a hydrocephalus internus, but still optic neuritis, terminating in atrophy, surpasses all other ocular symptoms in meningitis simplex. An amaurosis of cerebral or cortical origin may occur in this form of meningitis, but frequently the vision cannot be improved, although the ophthalmoscopic condition is negative and the pupillary reactions are intact. Even when the meningitis is at the base of the brain and causes an optic neuritis with atrophic discoloration of the papilla, a portion of the vision may be preserved. All other ocular symptoms, such as pareses of the muscles of the eye, are rare in this form of meningitis, except that pareses of the abducens are fairly frequent. The importance of the eye symptoms is quite different in the other forms of this disease. A secondary meningitis may occur in the course of the general infectious diseases, such as typhoid fever, pneumonia, scarlet fever, influenza and pyæmia, but such cases are comparatively rare, so I will mention only the principal forms—tuberculous, epidemic cerebrospinal, and otogenous purulent meningitis—in each of which the ocular symptoms have a certain degree of diagnostic importance.

I. Tuberculous Meningitis

If it is suspected that a child has tuberculous meningitis we ascertain first whether an *optic neuritis* is present, for such an inflammation of the optic nerve, characterized by redness and haziness of the tissue of the papilla with obscuration of its margins without a great degree of swelling, is the *most common ocular symptom of tuberculous meningitis.* It is present in from 25 to 30% of the cases.

A prominent choked disc is much more uncommon. Such a condition, especially when it is bilateral and met with in the course of a meningitis, must lead us to suspect a complication, either by a solitary tubercle, or by hydrocephalus.

Descending neuritis with great loss of vision and slight changes in the fundus is equally rare. Simple hyperæmia of the papilla and hemorrhages into the retina are infrequent in tuberculous meningitis and have little diagnostic value.

We meet with discrete tubercles of the chorioid rather more often, according to *Uhthoff* in about 10% of the cases. These form little, yellowish gray, distinctly prominent and usually sharply defined foci in the chorioid, and have a predilection for the region of the posterior pole. They may occur together with a neuritis, or independently, but they are much more common in general miliary tuberculosis, in which they are found in nearly half of the cases. Taking all these lesions of the optic nerve and chorioid together, neuritis, choked disc, hyperæmia, descending neuritis and tubercle of the chorioid, we find, according to *Uhthoff*, pathological changes in the fundus in 49% of the cases of tuberculous meningitis.

As regards affections of the ocular muscles, oculomotor paresis is much more frequent in gummatous meningitis of the base of the brain, and a bilateral paresis of this nerve is scarcely to be met with. A conjugate deviation of the eyes is very rare, and nystagmus is by no means frequent.

Impairment or complete abolition of the reflex reaction of the pupil to light is to be observed in from 10 to 15% of the cases. A difference in the size of the pupils is about as frequent. The principal ocular symptoms in tuberculous meningitis are to be detected with the ophthalmoscope, and by their help we can often render positive the differential diagnosis from other acute infectious diseases with cerebral symptoms. A marked neuritic optic atrophy usually indicates that the meningitis was not tuberculous, because death generally intervenes before such a condition can develop. Hemianopic defects in the visual field are scarcely ever observed in this disease.

II. Epidemic Cerebrospinal Meningitis

In this form also optic neuritis takes first place among the ocular symptoms, while choked disc is very rare. Retinal hemorrhages are uncommon. Even when no lesions can be seen in the fundus with the ophthalmoscope visual troubles may occur which are to be ascribed to lesions situated either at the base of the brain, or in the cortex of the occipital lobe. These may be differentiated by means of the pupillary reactions. When the amaurosis is absolute and the reaction of the pupil to light is still normal the lesion cannot be situated at the base of the brain or peripherally.

Troubles with the muscles of the eye are met with in from 13 to 15% of the cases, according to *Uhthoff* and *Heine*. The paresis of the abducens is the most common, that of the oculomotorius is less frequent. The other troubles, such as strabismus, nystagmus, and conjugate deviation, are of no great importance to the diagnosis. Pupillary disturbances are rarely met with, and *Leichtenstern* maintains that an extreme dilatation of the pupil with loss of its reactivity is far more frequent in tuberculous than in epidemic meningitis. The palpebral fissures gape widely, and winking is diminished in deep stupor, but this lagophthalmos is not the result of a facial paresis, it is due to the stupor of the sensorium.

Inflammations in the orbit are very rarely met with. Neuroparalytic keratitis is no more apt to occur in epidemic than in tuberculous meningitis, but the two diseases may sometimes be differentiated by the fact that a metastatic ophthalmia takes place in about 5% of the cases of epidemic cerebrospinal meningitis, either in the early or in the late part of the course of the disease. A very remarkable fact is that this symptom may appear even in very mild cases, and may be so slight that the eye may regain something of vision. This sort of metastatic ophthalmia begins with grave inflammatory symptoms which quickly retrograde, and the eyeball atrophies only a little. Such a benign form is excited only by pneumococci and the phenomenon is explained in cerebrospinal meningitis by the fact that meningococci die very soon in the vitreous. The metastatic ophthalmia may start either in the vessels of the retina, or in those of the chorioid, but it must not be thought that the inflammation is transmitted along the optic nerve from the meninges; it is of hæmatogenous origin. Although this ophthalmia runs on the whole a benign course, so far as the eye is concerned, its appearance is of little value to the general prognosis as to life, for the patients may die just the same.

III. Otogenous Purulent Meningitis

A large proportion of the cases of purulent meningitis are caused by diseases of the ear and of the petrous portion of the temporal bone, and the diagnostic value of the ocular symptoms produced in this disease is quite different from that in the forms of meningitis that have been mentioned. Metastatic ophthalmia need not be taken into account, as it is met with in practice very rarely. Optic neuritis is uncommon in the absence of complications; when present it indicates a circumscribed meningitis and that the otologist must be called upon for an operative intervention. A typical choked disc usually indicates other intracranial complications, such as abscess of the brain, or sinus phlebitis.

The abducens is the most frequently affected of all the nerves of the ocular muscles; it runs over the apex of the petrous portion of the temporal bone and may readily be involved in a circumscribed meningitis. Next in frequency the facial nerve is affected, but troubles of the other nerves are rare. We cannot learn much from the behavior of the pupils, and on the whole it may be said that ophthalmoscopic changes are not very common in otitic meningitis. Abscess of the brain is the complication in which optic neuritis or choked disc is most often met with.

When it is suspected that a cerebral hemorrhage, a hæmatoma of the dura, is present in a case of *pachymeningitis interna hæmorrhagica* we look for a choked disc or an optic neuritis, for it has been ascertained that in such hemorrhages the blood enters the sheath of the optic nerve and excites the symptoms of engorgement of the papilla. The diagnostic value of choked disc in only one eye is particularly great, for experience has taught us that such a condition is very rare in such other troubles as tumor of the brain, abscess, hydrocephalus, and even in fractures of the base of the skull and lacerations of the middle meningeal artery. Retinal hemorrhages are also found in many cases of pachymeningitis interna hæmorrhagica. The other ocular symptoms are of slight diagnostic importance in a hæmatoma of the dura in comparison with this condition of the optic nerve. Pareses of the ocular muscles are very rare. A conjugate deviation of the eyes to one side, commonly with a rotation of the head to the same side, is the most positive symptom. Nothing diagnostic can be obtained from the nystagmus, or from the anomalies of the pupils.

165. Ocular Symptoms in Hydrocephalus

The diagnosis of hydrocephalus is not difficult when a child develops after birth an increasing size of the head to which the parents themselves call attention. In this case we have not simply to make the diagnosis, but to pass judgment upon it ophthalmologically, for our colleague, *Payr*, is about to undertake its operative treatment, and the ophthalmoscopic control of the intraocular changes is one of the preconditions to the adoption of all therapeutic attempts in hydrocephalus.

Whether this condition starts as a serous meningitis, or from an occlusion of the aquæductus *Sylvii*, the result is always an abnormal accumulation of fluid in the cerebral ventricles, and, in rare cases, between the dura and the surface of the brain as well, which is associated with an increase of the intracranial pressure.

In this child we find the most common ophthalmoscopic symptom of the disease, a choked disc in each eye. Rather less frequently an optic neuritis, or a neuritic optic atrophy is met with in hydrocephalus internus, which is indicated by such a condition, as has been proved by observations confirmed by autopsy. Such changes in the optic nerve are almost never observed in hydrocephalus externus.

A fact to which I wish to call particular attention is that we very often meet with a simple optic atrophy in chronic hydrocephalus without any signs of a preceding inflammation. This simple optic atrophy is found in the smallest children in congenital hydrocephalus, and is considered by most ophthalmologists to be a pressure atrophy caused by the pressure exerted by the bulging floor of the third ventricle upon the optic tract.

It happens not so very rarely that the vision is found to be lost or greatly impaired without any cause being visible in the fundus, especially in congenital hydrocephalus. It is supposed that this is due to pressure upon the optic paths of conduction at the base of the brain. Such a high degree of degeneration of the cerebral hemispheres was found in the cases in which I have witnessed the autopsies that the amaurosis had to be considered of cortical origin. Both factors, pressure on the paths of conduction and destruction of the cortex, evidently take part in the impairment of vision.

Troubles with the muscles of the eye are rare in hydrocephalus; paresis of the abducens is the most frequent. Pareses of the associated movements of the eyes are also met with. It has been observed repeatedly that the eyes are constantly turned down and cannot be moved upward, and such a position is certainly suggestive of this condition. In the course of a chronic or congenital hydrocephalus in children the upper wall of the orbit may be pressed downward by the increased pressure within the skull, so as to produce an exophthalmos which differs from that caused by disease within the orbit; the eyeball is driven forward more or less uniformly by the latter, but when the upper wall of the orbit gives way in hydrocephalus the eyeball is driven downward and forward. The roof of the orbit may be so thin that the distended dura may bulge directly into the orbital cavity. All of these conditions are to be met with only in very early life, before the bones cease to yield to the intracerebral pressure. Other deviations of the eyes are less frequent, except that nystagmus is seen rather often. The latter is usually a pressure symptom, but *Oppenheim* has pointed out that it may be caused by an affection of the labyrinth. Finally it may be mentioned that chronic hydrocephalus, even when uncomplicated, may cause such apparently focal symptoms as nystagmus, pareses of the muscles of the eyes, and of the facial nerve, cerebellar ataxia and tremors that come on when efforts at voluntary motion are made (Intentionstremor).

When we come to the question as to what ocular symptoms may be induced to retrogress by operative measures we find that we can follow the height of the choked disc very accurately. No involution of a neuritic or genuine atrophy can be expected. As the signs of cerebral pressure pass away we may be able to observe an improvement in the derangement of the muscles of the eyes, especially of the nystagmus.

166. The Great Diagnostic Value of the Ocular Symptoms in Tabes

This patient has a commencing simple atrophy of both optic nerves and a reflex immobility of his pupils. The etiology is indicated by the absence of the patellar reflex. The report of the neurologist is as follows: E. S., 30 years old, states that he first noticed an impairment of his vision four weeks ago. Has always been in good health. Family history negative. Had a venereal disease which may have been gonorrhœa three years ago. Has often had opportunities to catch cold. Gave up drinking during the past year. Has no trouble with the bladder. No crises, no typical lancinating pains, but of late has sometimes had jerkings of the legs. No paræsthesia. Tongue protrudes straight forward, trembles slightly and is furrowed. Movements of tongue free. Facial nerve and act of swallowing normal. Movements of eyes free. Corneal reflex +. Muscles of limbs and body strong. Slight wavering when standing with the eyes closed. No ataxia. Sensation for change of position good. Stands badly on one leg. Distinct hypalgesia on the thorax above and below the mammæ as high as the clavicle. The ulnar portion of the forearm and hand are the less sensitive to pain. Tactile sensation good. Patellar reflex absent on both sides. Achilles tendon phenomenon not to be excited. All skin reflexes +. No *Babinski*. Tendon reflexes of the upper extremity absent. Pressure on the deep muscles of the nape of the neck painful. Diagnosis: Tabes.

The ocular symptoms play an important part in making the diagnosis. Three of these are of special interest; the reflex immobility of the pupils, which is a most common symptom in this disease, the troubles with the ocular muscles which come next in frequency, and the simple optic atrophy.

I. Ocular Symptoms of Tabes

Reflex Immobility of the Pupils

Argyll-Robertson, of Edinburgh, was the first to call attention to the great diagnostic importance of this symptom in nervous diseases, which is so great that the pupils should be examined first of all when suspicion arises that a patient has tabes. The figures given to indicate the frequency with which reflex immobility of the pupil is met with in tabes vary according to the stage of the disease and the clinical material, but we may say that it is present in almost 75% of the cases. Many claim it to be even more frequent than the absence of the patellar reflex. The fact is important that it is demonstrable in most cases in the preatactic stage and is therefore a valuable early symptom. Usually it affects both eyes at the same time, but it has been observed in one eye alone on rare occasions and the point should be emphasized that the diagnostic significance in such a case is the same as when the immobility is present in both eyes. We speak of a reflex immobility of one pupil when light excites no direct reaction in it, but does excite a consensual reaction of the other, while illumination of the second eye produces a direct reaction, but not a consensual.

A satisfactory explanation of the occurrence of reflex immobility of one pupil alone has not yet been obtained. Some investigators, like *Moebius* and *Schwartz*, ascribe it to a unilateral lesion of the centripetal reflex tracts after their separation from the visual fibres, between the corpus geniculatum and the nucleus of the sphincter pupillæ. Others think it due to a lesion of the centrifugal fibres peripherally from the nucleus of the sphincter. Others again believe it indicates a nuclear lesion. The latter start from the assumption that there are two groups of ganglion cells in the nucleus of the sphincter, one of which is responsible for the transformation of the light stimulus into iris movement, while the second governs the other stimulations of the sphincter. But the entire question as to the anatomical explanation of the reflex immobility of the pupil is still unsettled. Changes in the central tubular gray matter of the third ventricle and in the aquæduct of *Sylvius* have been found in some of the cases that have come to autopsy, and the reflex immobility of the pupil has been ascribed to a disturbance of the reflex arch between the optic and the oculomotor nerves on the basis of such findings, i.e., to a lesion of the light conducting fibres close to the nucleus of the sphincter. On the other hand there are neurologists who locate the cause in the cervical portion of the spinal cord. Thus *Rieger*, *v. Forster*, *Wolff* and *Bach* place it in the posterior columns of the upper part of the cervical spinal cord. At all events its cause is not to be found in a primary degeneration of the pupillary fibres of the optic nerve, for the retina and nerve may remain perfectly normal, both ophthalmoscopically and anatomically, after a reflex immobility of the pupil has existed for a long time. It must be left to future research to discover the site of the lesion.

We have next to determine whether the immobility of the pupil is total, or whether the reaction to convergence is preserved. It happens very often in tabes that the pupil continues to react to convergence until the last stages of the disease, although the direct reaction to light is absent, and when the reaction to convergence is not present it is usually true that the reaction to light was lost long before. The reaction to light has been observed to return for a time in rare cases, but such a phenomenon, which is met with much more often in cerebral syphilis, seems to be extremely infrequent, and the rule is that when the reaction to light has once been lost in tabes it never returns.

The so called recovery reaction of the pupil has no great clinical significance. In these cases the reaction to light improves after the patient has remained in the dark for a long time.

The myotonic pupillary movement is the name given to the phenomenon found by *Saenger* in tabes, that a pupil immobile to light, but reacting to convergence, remains contracted for several minutes after the act of convergence.

The reaction of the pupil to sensory stimuli needs to be tested accurately only in rare cases, for we know from the researches of *Erb* that the pupil does not dilate in response to sensory stimuli when it is in a state of tabetic reflex immobility.

It has been said that the *Argyll-Robertson* pupil is a sure sign of former syphilis, but this is not true. Syphilis causes it occasionally, sometimes, though rarely, at an early period, but cases of tabes with reflex immobility of the pupils are met with in which there has been no syphilitic infection. One fact however will strike every clinician, that such complications as atrophy of the optic nerve and paresis of the muscles of the eye are apt to be more frequent in those cases of tabes that have been preceded by syphilis.

Meiosis may appear very early in tabes. I know a man whose meiosis was extreme for twenty years before another symptom was demonstrable. If we consider meiosis as pathological only when the diameter of the pupil is at most 1.5 mm we will find it combined with reflex immobility of the pupil in about 25% of the cases. It is of spinal origin and due to a disturbance of the innervation to dilate the pupil. The centrum ciliospinale (*Budge*), in which the lesion is usually situated, lies at the level of the last cervical and the first dorsal vertebræ. Some do not believe in this sympathetic theory, but ascribe the meiosis to an irritative condition of the centripetal reflex fibres (*Heddæus*). Meiosis and reflex immobility of the pupil may occur separately, so we suppose with *Erb* that they come from two separate lesions.

Great weight has always been laid on anisocoria of the pupils, but to value this symptom rightly we must remember that an inequality of the pupils is met with in healthy persons. In such a case the reaction to light will be good. Differences in the sizes of the pupils are also met with in other organic diseases, especially in those of the thoracic organs. Of the diseases of the central nervous system general paralysis is the one in which a difference in the size of the pupils is most common. Anisocoria is present in about 25% of the cases of tabes, when it is usually accompanied by a reflex immobility of the pupil. A diagnostic importance can therefore be ascribed to an inequality of the pupils only when the reaction is impaired at the same time.

A so called jumping mydriasis has been observed in this anisocoria in rare cases of tabes, in which sometimes one, sometimes the other pupil is the larger, but this phenomenon may occur in hysteria, as well as in quite a number of other general diseases.

The so called paradoxical pupillary reaction, in which the pupils dilate in response to light, is extremely rare in tabes. Frequently the sphincter contracts only a little, and then the pupil dilates in spite of the entrance of light into the eye. It should also be noticed whether the dilatation of the pupil is not a movement that accompanies changes in the accommodation, and in the position of the eye. We must first exclude this apparently paradoxical reaction whenever we think we have a case of paradoxical movement of the pupil.

When I hold the lids of this patient apart and direct him to close his eyes a contraction of the pupil may be seen. This is the reaction to the closure of the lids. It is usually present when the reaction to convergence is preserved in a pupil with tabetic reflex immobility, but cases of tabes have been met with in which either the reaction to convergence, or that to the closure of the lids is alone preserved intact.

II. Ocular Symptoms of Tabes

Paresis of the Muscles of the Eye

An impairment of the movements of the muscles of the eye is by no means as frequent as the reflex immobility of the pupil, to which it ranks second as an important ocular symptom. According to *Uhthoff* these pareses occur in 20% of the cases of tabes, and 20% of the cases of pareses of the ocular muscles are due to tabes. Every fifth tabetic has a paresis of the ocular muscles, and every fifth patient with such a paresis has tabes. We are coming more and more to believe that the danger of trouble with the muscles of the eye is greatest in those paretics who have suffered from syphilis.

Tabetic pareses of the ocular muscles have several characteristic clinical features. As a rule they appear relatively early in the course of the disease, they have a fleeting character, are partial and often pass away quickly. They also tend to recur. The same muscle may be attacked several times, or, when the first attack is over, the paresis may seize upon some other muscle. Partial pareses of the oculomotor nerve are the most frequent, the abducens is affected less often and the trochlearis very rarely. It is remarkable that ophthalmoplegia interna seldom occurs in tabes. The usual cause of both bilateral paresis of the entire oculomotor nerve and ophthalmoplegia interna is syphilis. On the other hand ophthalmoplegia externa is occasionally met with in the former disease. *Struempel* considers the changes in the peripheral nerves of the ocular muscles to be responsible for the transient pareses, but the majority of tabetic affections of this nature are caused by lesions in the nuclei. The ganglion cells are pathologically altered and degenerative changes take place in the fibres in the trunk of the nerve that supply the muscles.

Nystagmus is very uncommon, and when we meet with it we suspect the presence of other cerebral complications, such as multiple sclerosis, or that it is congenital. Twitchings of the eye that resemble nystagmus when the eyes are turned as far as possible in one direction are observed rather more frequently.

In rare cases of tabes sensory disturbances occur in the region of the trigeminus which may give rise to a sense of constriction in the face, *Charcot's* tabes mask, but such neuralgic pains are infinitely more rare than the well known lancinating pains in the body. It is an almost characteristic feature of this disease that the trigeminus and facialis are seldom affected, and in the rare cases in which increased lacrymation has been observed it is questionable whether there has been a lesion of these nerves.

III. Ocular Symptoms of Tabes

Atrophy of the Optic Nerve

Atrophy of the optic nerve occurs in about 10% of the cases of tabes and may appear at any stage of the disease. Although it may be observed in the ataxic and paralytic stages its onset is most frequent in the preataxic stage, and it may precede all other symptoms of the disease by several years, a fact which is of considerable diagnostic importance.

This atrophy was believed for a long time to indicate a good prognosis for the tabes, and it is a fact that we find the well defined symptoms of ataxia in only a small percentage of our cases with atrophy of the optic nerve. Many writers have claimed that the onset of optic atrophy indicates a relatively slow course of the tabes. Tabes superior, ocular tabes, benign forms of tabes, have been spoken of, but we can in no way depend on this in individual cases, for the better the neurological methods of investigation have become developed the more it has been shown that even in cases of ocular tabes symptoms appear which show the lesion to be in the lower segment of the spinal cord, such as absence of the patellar reflex, paræsthesias of the legs, sensory disturbances of the intercostal nerves, and troubles with the bladder.

The question in which the patient himself is most interested is the course of the visual trouble, which consists of a diminution of the central vision with a progressive contraction of the visual fields. These two troubles begin very gradually and go hand in hand. The patients complain that the objects seen become paler and more indistinct. Subjective signs of irritation, such as dazzling and photopsias, are rare. One eye is ordinarily affected before the other, and it may happen that the blindness of the first eye is not noticed until after the second has begun to fail; but sometimes both eyes suffer uniformly. The patients lose their power of color perception as their central vision fails. The green leaves on the trees seem to them more and more colorless, red can no longer be recognized, and so on. The central vision continues to fail, the peripheral vision to contract more and more, until the result is total amaurosis. *The course of the disease until amaurosis is complete requires from two to four years on the average.* Occasionally the time is shorter, in very fortunate cases it is longer, from six to ten years, and exceptional ones have been met with in which the vision has been retained till the end of life.

The nature of the visual disturbance can be appreciated from a study of many visual fields obtained from various patients, which reveal two fundamental types of contraction. When the entire optic nerve is affected from the first a concentric contraction of the visual field from the periphery takes place coincidently with the lowering of the central vision. The power of distinguishing red and green is lost first, while white objects can still be correctly perceived within the normal limits. This impairment of the color perception at the beginning of the disease is of great diagnostic importance. The limits for white then contract gradually, then the power of distinguishing blue from yellow is lost; blue appears more and more like gray, and finally the color blindness is total in what remains of the field for white. The more intensive the colors and the brighter the illumination the longer can the colors be recognized, a point of differentiation between this and the congenital form of color blindness. Finally the field becomes so small that it cannot be tested.

In another group of cases the contraction of the visual fields may be in the form of sectors, or it may be concentric and of high degree when the central vision and color sense seem to be preserved for a longer time, but the contractions of the other segments of the field always follow in a short time, and the atrophy of tabes differs from that of the papillomacular bundle of optic nerve fibres productive of a central scotoma in that the functional disturbances in the periphery of the field are never absent. As the condition in tabes is one of degeneration of the retina commencing at the periphery, it can give rise to no homonymous or heteronymous hemianopsias, unless such happen to be produced by other cerebral diseases, but sometimes the fields remind one of hemianopsia, when the contraction in the two eyes is uniform and begins on the temporal side.

Hemeralopia is absent, the eyes are, on the contrary, rather sensitive to bright light, so that patients sometimes think they can see a little better in the dusk.

Tabetic atrophy of the optic nerve begins in the peripheral portions of the optic path of conduction, probably in the layer of ganglion cells and the layer of nerve fibres of the retina. This change cannot be seen with the ophthalmoscope, the first sign visible in this way is the atrophy of the papilla. *Wagenmann* was able to observe directly the atrophy of the medullary sheaths in the retina in a tabetic patient who had medullated nerve fibres. We can infer clinically that the atrophy commences in the periphery of the optic path of conduction from the fact that the visual disturbances begin with the atrophy of the papilla, and that the latter may precede the former. It is generally acknowledged at the present time that the degeneration of the optic nerve fibres in the retina and optic nerve is the primary symptom, while the changes in the interstitial tissue and the neuroglia are thought to be secondary. The medullary sheaths in the optic nerve break down, irregular constrictions appear in them and they finally disappear. The axis cylinders become varicose in places, thinned elsewhere, and are destroyed. This process results in the appearance of products of destruction, such as fatty granular globules and drops of myelin. Proliferations in the interstitial tissue are absolutely wanting in this first stage, but later the connective tissue septa appear to have become broadened and have a homogeneous appearance without the presence of any inflammatory cellular infiltration and proliferation of nuclei. The fibres of neuroglia and their nuclei seem to be increased, but still the structure of the optic nerve with its septa and meshes can be recognized on the slide. The purely atrophic changes in the layer of nerve fibres and of the ganglion cells in the retina are also unaccompanied by any proliferative process. The increase of neuroglia in tabetic atrophy is, according to *Weigert*, secondary to the irritation produced by the degenerating nerve fibres. Why there should be a certain predilection for the atrophy to affect the optic nerve in tabes still surpasses our knowledge. Many hypotheses have been advanced, but a consideration of them would take us too far afield. They are all finally lost in the dark problem of predisposition.

One thing is certain. Syphilis plays an important part in tabetic atrophy. The connection between these two diseases has been accepted by many writers, like *Erb* and *Moebius*, and the proportion in which syphilis is present in tabetic atrophy may be estimated as from 60 to 80%. *Wassermann's* reaction shows scarcely one that is not syphilitic. The statement of *Uhthoff* seems to me to be very true that "those who have not had syphilis have every chance never to acquire a progressive tabetic atrophy." Although the connection between these two diseases is positive there is nothing certain about the time of the onset of the atrophy after the luetic infection. According to the statistics I have compiled, several years, from four to six on the average, must elapse before the degeneration of the optic nerve is brought about, and in the overwhelming majority of cases many years intervene between the infection and the onset of the tabetic atrophy.

It is very difficult to estimate the influence of treatment, because the course of a tabetic atrophy of the optic nerve is very variable, even without therapeusis. In rare cases the process may come to a standstill; in other cases just as rare the destruction is confined to only one nerve, and the time which elapses before the onset of the amaurosis varies so much that we cannot tell how much it is affected by treatment. Hence there remains nothing else for us to do than to try to arrest its development by the proper treatment of the tabes itself. Because of the connection between syphilis and the optic atrophy the question naturally arises whether a mercurial treatment should not be instituted, and we would certainly answer this in the affirmative were it not that sometimes both the tabes and the optic atrophy are made worse by such treatment, so that it must be used with great care. This treatment is indicated when the differential diagnosis between tabes and cerebral syphilis cannot be made in a commencing optic atrophy. We must also, according to *Erb*, treat those tabetics with mercury in whom the infection occurred a short time before the onset of the atrophy, or in whom the syphilis has not been subjected to a proper treatment.

167. The Limited Diagnostic Value of the Ocular Symptoms in the Other Diseases of the Columns and Systems of Fibres of the Spinal Cord

Diseases of the spinal cord may be divided into two large groups: those of the columns and of certain systems of fibres, and those that are more diffuse. Clinical experience has shown that there is one disease in each of these groups in which the ocular symptoms are of great importance: tabes in the first, multiple sclerosis in the second. The value of the ocular symptoms in tabes has been shown, but their diagnostic importance in the other diseases of the columns and systems of fibres of the spinal cord is much less, in some they are negative. Quite a number of diseases have to be taken into account: Hereditary ataxia (*Friedreich's* disease), spastic spinal paralysis, combined disease of the posterior and lateral columns, poliomyelitis anterior acuta and chronica, acute ascending spinal paralysis, and the primary degeneration of the motor paths of conduction.

In hereditary ataxia the only reliable and constant ocular symptom is the irregular twitching of the eyes, that resembles nystagmus, when they are turned to one side. This is a very rare disease, and when it needs to be differentiated from tabes it may be noted that this nystagmus does not occur in the latter disease, and that the derangements in the optic nerve and pupil are not found in the former.

Ocular symptoms are wholly wanting in spastic spinal paralysis, which is rare, and is due to an isolated degeneration of the lateral columns of the spinal cord.

The Combined Disease of the Posterior and Lateral Columns of the Spinal Cord

The ocular symptoms found are quite analogous to those met with in tabes, simple optic atrophy, pareses of the muscles of the eye, and immobility of the pupil, yet they occur more rarely and increase in proportion to the degree in which the degeneration of the posterior columns appears prominent in the combined disease.

Congenital Spastic Rigidity of the Limbs, Little's Disease

A concomitant strabismus is present in from 25 to 30% of the cases of congenital or early acquired spastic paralysis, and forms the most important and most frequent of the ocular symptoms of this disease. It is supposed to be caused by tonic spasms of the ocular muscles. Nystagmus and atrophy of the optic nerve occur very rarely indeed, in comparison with this symptom, which is really the only one to be taken into account in making the diagnosis.

Poliomyelitis Anterior Acuta et Chronica

No ocular symptoms that have any diagnostic significance occur in either of these diseases, which are characterized by a degeneration of the gray substance of the anterior cornua.

Acute Ascending Spinal Paralysis (Landry's Paralysis)

Here we occasionally meet with pareses of the ocular muscles, usually of the abducens, because its nucleus is the lowest. Disturbances of the pupil and optic nerve are rare.

Primary Degeneration of the Motor Paths of Conduction

The nerves of the muscles of the eye are seldom involved in amyotrophic lateral sclerosis, and the other ocular symptoms have as little diagnostic importance. They likewise have no special diagnostic value in the other diseases belonging to this group, characterized by atrophy of the muscles, among which may be named progressive muscular atrophy, neurotic muscular atrophy, and primary myopathy, which is also known as progressive muscular dystrophy. In this latter disease the degenerative paresis of the orbicularis, which causes an imperfect closure of the lids, alone deserves our attention. The muscles of the eye itself are very seldom involved.

As I have said, the only disease in this entire group in which the ocular symptoms are diagnostic is tabes. The conditions in the second group, which includes the more diffuse diseases of the spinal cord, are quite analogous. The ocular symptoms are characteristic in multiple sclerosis, but are of little diagnostic value in the other diseases.

168. The Great Diagnostic Importance of the Ocular Symptoms in Multiple Sclerosis

The history of this patient is as follows: A. R., 29 years old. Father is very nervous, mother is in good health. Eight out of twelve brothers and sisters died when very young, none with convulsions. Two relatives on his father's side are blind. The patient had attacks of stupor when a child and again when he was 18 years old. As a soldier in 1898, at 20 years of age, he was perfectly healthy, the strongest man in his company and a good shot. In 1902 frequent attacks of stupor again appeared. In 1905, when returning quite sober from a ball, he suddenly fell without any apparent cause, but was able to get up and go on at once. In January, 1906, he made himself independent under great difficulties; he frequently worked all night, ate badly and had a great deal of excitement in the way of business. One day in March, 1906, he noticed that he could not see well centrally with his left eye, although its peripheral vision was good. He consulted a physician, who prescribed iodide and said the disease was an inflammation of the optic nerve behind the eye. He is unable to give further details as to whether the visual disturbance persisted a long time or not, because he saw well with his right eye and this sufficed for his purposes. He was so absorbed in his business cares that he neglected everything else. In January, 1907, after a period of great mental excitement, he suddenly saw a cloud before his left eye, which disappeared in half an hour, but returned frequently after vexation and excitement and increased in duration. He says he was able to check it by dousing his head in cold water. The physician who was finally consulted prescribed aspirin, sajodin and vapor baths. Attacks of vomiting and dizziness followed the latter and persisted after the baths had been discontinued. One morning near the end of August, 1907, after a great business vexation the preceding evening, he noticed that the central vision of his right eye was gone. From this time on the attacks of dizziness became more frequent, he suffered from headaches and sometimes from dull pains behind the eyes. In addition he could not walk well at times and sometimes fell without apparent cause. As his condition continued to grow worse he sold out his business in September, and in November as he was looking over the account he was struck with total paralysis of his right leg and partial paralysis of his right arm and hand. After that, in spite of a feeling of tension in his bladder he was unable to void his urine properly. During this time his vision grew considerably worse. The paralysis improved gradually, so that at the beginning of December he could use his hand and arm and could walk with the aid of two canes. The weakness of the detrusor muscles of the bladder also improved, but the badly impaired vision showed no signs of recovery, and this brought him to us on the 3d of March. He has used alcohol and tobacco very moderately and denies that he has ever had syphilis.

The vision of his right eye is 2.5/60, that of his left 3/60, not improved with glasses. He cannot read the largest print. The skiascope shows that he has 1 D of hypermetropia in his right eye, 0.5 D in his left. The direct and consensual reactions to light are good in both eyes, that to accommodation is almost lost. The motility is unimpaired. He has a slight nystagmus of both eyes.

Fundus: The temporal side of each papilla is very characteristically pale, while the nasal halves have their normal yellowish red color. The stippling of the lamina cribrosa is distinctly visible in the pale portion. The margins of the nerve are sharply defined, especially on the temporal side. The larger vessels are well preserved but contracted, the arteries more so than the veins. No lesion is to be seen in either macula.

Field of vision: Moderate concentric contraction of the right field for white and blue; no contraction above and below, a slight contraction on the nasal side, a greater one on the temporal, where the limits for white are rather irregular. The limit for red is contracted below the horizontal line to 20°, is less contracted everywhere else, and at one point it projects over that for blue. The limit for green is distinctly contracted concentrically. In peripheral vision blue is correctly recognized with each eye, red appears brown, green appears yellowish. He has an absolute central scotoma for green, extending upward and outward to 15°, below to 20°, inward to 12°, and an absolute scotoma for red situated paracentrally in the region of the blind spot about 5° in diameter. In the left eye we find an absolute scotoma for green extending about 12° in all directions, and another for red extending upward, inward and downward 10°, outward 5°. He has an ill defined relative scotoma for blue, sometimes central, sometimes paracentral to the nasal side, varying in size, in which blue appears to be gray. The white point of fixation on the perimeter often vanishes for seconds before each eye of the patient and its size seems to change. Nothing pathological was revealed by the examination of his urine.

Neurological examination: The right leg is slightly spastic in walking, the sole of the foot drags along the floor. Uncertainty in turning around, not made worse by closing the eyes. Stands badly on one leg, especially the right. Strength preserved everywhere. In the fatigue test vacillations are noticeable on the right side in twenty seconds, though the leg can be held up a minute without difficulty. Slight spasms greater on the right side than on the left. Impossible to arise from lying on the back. Slight uncertainty of the hands in the test of touching the nose with the finger. No ataxia in the legs. The sense of change of position preserved everywhere. All qualities of sensation good, except that perhaps the feeling of touches of a pencil on the right foot may be a little weak. All tendon reflexes increased. He has ankle clonus and clonus of the patella. *Babinski* greater on the right side than on the left. Abdominal reflex apparently absent on both sides. Reflex of the frontal branch of the right facial nerve less than that of the left. Associated movements about the mouth when the tongue is protruded. Movements of the tongue, of mastication and of swallowing free. Masseteric reflex not increased, cremasteric reflex weak on both sides. Slight ptosis of right upper lid. Pharyngeal reflex +. Marked tremor and *Quinquaud.* Speech slow but distinct. The serological examination for syphilis gives a negative result. Diagnosis: Multiple sclerosis.

The diagnosis of multiple sclerosis is not always so simple and clear, but the symptoms presented by the eyes often need to be taken into account in the early stage of this disease. The most common ocular symptom in tabes is the reflex immobility of the pupil, then come pareses of the muscles and, last, the signs of atrophy of the optic nerve. The condition is exactly reversed in multiple sclerosis, in which the changes in the optic nerve and the disturbances of vision take first place, the troubles of the ocular muscles second, and the changes in the pupil last of all.

I. Changes in the Optic Nerve and Disturbances of Vision

In many cases the picture presented is that of *simple atrophy,* such as we are accustomed to meet with in tabes. The papilla is completely atrophic, its margins are sharply defined. Frequently a slightly reddish color can be seen in the inner half of the papilla, but I consider it an affectation to make two subdivisions of the simple optic atrophy that we meet with in multiple sclerosis.

Just as often we find the *atrophy confined to the temporal half of the papilla,* an ophthalmoscopic picture that is also to be seen in alcohol and tobacco amblyopia. In comparison with tabes *optic neuritis* is frequently met with in multiple sclerosis, though in a much smaller percentage of cases than the atrophic changes. I cannot venture to decide whether these cases are really more common than they appear to be, whether it is true that in multiple sclerosis an original optic neuritis may pass over into a simple atrophy, but in my cases the margins of the papilla have been rather indistinct.

Another clinical feature of this disease is that for a long time only one optic nerve may be affected, a fact that has to be taken into account in the individual cases, but we shall not learn the entire significance of the changes in the optic nerve as regards the diagnosis of multiple sclerosis until we ask: How frequently are these changes and the disturbances of vision present in this disease?

Significance of the Changes in the Optic Nerve and of the Visual Troubles in the Diagnosis of Multiple Sclerosis

The researches of *Uhthoff* have shown that the changes in the optic tract are present in about one half of the cases, and stress has rightly been laid on the fact that such a high percentage of pathological conditions in the optic nerve is exceeded only by tumors of the brain, and equalled only by cerebral syphilis and tuberculous meningitis. The diagnostic importance of these symptoms does not depend solely on their frequency, but also on the fact that they may precede the others. Visual troubles are present in a good many cases when we are unable to detect the cause with the ophthalmoscope, and they often furnish certain data for the diagnosis.

I called attention to the fact that in tabes, when we can see an atrophic paleness of the papilla, there is a corresponding impairment of function, but this relation between the visual disturbances and the ophthalmoscopic change is by no means so constant in multiple sclerosis. We may find an atrophic paleness, or an optic neuritis, and yet the vision may be as good as could be desired, or the function of vision may be considerably impaired without any corresponding ophthalmoscopic change being perceptible. In other cases the two factors go hand in hand. We must bear in mind that in multiple sclerosis the changes in the head of the optic nerve need not indicate the extent or the degree of the anatomical lesions in the nerve itself.

The impairment of vision develops quite gradually in tabes, while in multiple sclerosis it frequently comes on much more tempestuously, sometimes quite suddenly. When the onset of the disease is sudden the visual trouble often goes on to blindness, but in certainly half of the cases it takes place more slowly. The impairment of the vision is governed by no fixed rule.

The visual troubles of multiple sclerosis also differ in their course from those of tabes. Tabetic atrophy goes on to amaurosis in the overwhelming majority of cases, while in multiple sclerosis total blindness is an absolute rarity, and even in the most doubtful cases improvement is apt to take place and to be followed by a recurrence. These fluctuations in the visual disturbances form one of the characteristic features of this disease. The patients complain simply of indistinct or bad vision; they have no flashes of light or other subjective symptoms of irritation, so we can gather no data for the diagnosis from their descriptions. The examination of the field of vision furnishes us better information, although we are more or less impressed by a lack of rule in its behavior. We find central scotomata, with the periphery of the field free or contracted. Here again we meet with regular and irregular concentric contractions. Ring scotomata are seldom met with. Sometimes the various disturbances in the visual field change about, even within a short time.

When the visual disturbance takes the form of a central scotoma the differential diagnosis between multiple sclerosis and retrobulbar neuritis may be difficult, especially when the latter is not due to chronic intoxication, has a sudden onset and shows analogous fluctuations in intensity. It should be remembered in such a case that central scotomata are still more common in retrobulbar neuritis than in multiple sclerosis, and that in the latter disease the central scotoma is usually relative, while in acute retrobulbar neuritis it is ordinarily absolute. This difference does not appear in the amblyopia of chronic intoxication, but the latter is bilateral, as a rule, while in multiple sclerosis the trouble is apt to be confined to only one eye. As acute retrobulbar neuritis may also affect only one eye, it is readily understood that the differential diagnosis cannot always be made from the ocular symptoms alone in any given case. According to *Uhthoff*, multiple sclerosis is present in only 3% of all retrobulbar inflammations of the optic nerve, including amblyopia from intoxication. If the latter is excluded, multiple sclerosis is present in 8%. It is evident from this that from the ophthalmoscopic point of view the differential diagnosis of multiple sclerosis from retrobulbar neuritis is more difficult than from tabes.

Difficulties may arise in the same way when the visual trouble starts either with or without a retrobulbar neuritis and when it is a question of excluding a commencing myelitis. The course of such a case is decisive. In functional hysterical amblyopia the contraction of the visual field is more regular than it is apt to be in this disease.

All of the peculiarities of the visual disturbances in multiple sclerosis become comprehensible as soon as we study the pathological changes that take place in the optic nerve tracts. The question has not yet been decided whether the changes of the nervous elements, or those of the connective tissue, of the neuroglia and of the vessels are primary in this disease. We are inclined to the idea that the disease starts from the surrounding tissue and not from the nerve fibres, for the fact is established that the axis cylinders are spared for a long time in the foci of sclerosis in the optic nerves and lose only their medullary sheaths. In this the process differs essentially from that of progressive tabetic atrophy, in which both medullary sheaths and axis cylinders are destroyed. This fact corresponds with the clinical symptom which has been emphasized as the most important—the preservation of the visual function. In addition to this we meet with distinct signs of proliferation in the connective tissue framework and in the glia, which lead one to think that the process must start here. We find on our slides proliferation of the glia and thickening of the interstitial tissue, together with intact nerve tissue. The sudden onset of the visual disturbance likewise indicates an interstitial inflammation. We also find an increase of the vessels and changes in their walls in the vicinity of the foci. On the other hand signs of simple atrophy, degeneration of the nerve fibres and atrophy of the medullary sheaths may be present, so that both processes may sometimes be combined.

II. Disturbances of the Ocular Muscles

These consist, first, of pareses, second, of nystagmus, or twitchings of the eye that resemble nystagmus, and, next to the changes in the optic nerve, form the most important ocular symptom of multiple sclerosis.

According to *Uhthoff* the pareses occur in about 20% of the cases. They present two characteristic features. In the first place they are transient, often pass away quickly, and it is very seldom that a lasting paresis is met with, but they are governed in this respect by no fixed rule, and the diagnosis of multiple sclerosis cannot be made from a paresis of a muscle of the eye alone. The second peculiarity is that the clinical picture very often indicates a central lesion. Thus we find in this disease pareses of the associated movements of the eyes, or of convergence, well marked ophthalmoplegia externa, as well as partial pareses of the oculomotor nerve. The lesions are situated in the centres of the associated movements, or in the nuclei of the ocular muscles. In this respect there is a distinct difference between the disturbance of vision and that of the muscles of the eye in multiple sclerosis. The disturbances of the ocular movements are caused by central or cerebral lesions, those of the vision often by peripheral ones in the path of the optic nerve.

Pareses of the associated movements of the eyes are much more common in multiple sclerosis than in tabes. On the other hand total paresis of the oculomotor nerve is scarcely met with at all in the former, while it is frequent in the latter. The same is true of ophthalmoplegia interna, it occurs often in tabes, but hardly at all in multiple sclerosis.

Our patient exhibits still another eye symptom, a horizontal nystagmus.

We must differentiate carefully whether this is a true nystagmus, in which the eyes swing to and fro in accordance with the associated movements, or are twitchings that resemble nystagmus, which are accustomed to appear when the eyes are turned far in any direction. This derangement of the movements of the eyes is very common in multiple sclerosis. *Charcot* called attention to its importance, and held it to be analogous to the tremors that come on when efforts at voluntary motion are made, but the views in regard to its nature are very much divided. Some think it due to an anomaly of the tonicity of the muscles, some believe it due to an imbalance caused by insufficiency of certain groups of muscles, some consider it a symptom of fatigue, and some think that it is brought about by fitful contractions of the muscles. True nystagmus alone is of any significance. It is rarely met with in the other diseases of the central nervous system as compared with multiple sclerosis, in 12% of the cases of which *Uhthoff* found it to be present, while twitchings that resemble nystagmus have been met with in all possible kinds of diseases, for example in affections of the labyrinth, meningitis, hysteria, tabes, alcoholism, paralysis agitans, and diseases of the cerebral cortex, and *Schultze* has pointed out that we can even find them in persons who are enjoying perfect health.

III. Behavior of the Pupils in Multiple Sclerosis

Complete reflex immobility of the pupil and meiosis occur very seldom in this disease, a respect in which it presents a marked contrast to tabes, but occasionally mydriasis, a somewhat reduced reaction to light, and a difference in the size of the pupils are met with. The presence of an abnormally lively pupillary reflex has been pointed out by *Parinaud* and others, but we cannot do much diagnostically with all these symptoms, we must always take into consideration along with them the changes in the optic nerve and the disturbances of the ocular muscles. The order of importance of the ocular symptoms in this disease is, first, visual troubles with the changes in the optic nerve; second, pareses of the ocular muscles and nystagmus; last, the changes in the pupil.

Therapeutically we are confined to the general treatment of multiple sclerosis: Electricity, hydrotherapy, massage, treatment of the ataxia by exercise. The most useful drugs are the iodides, mercury and arsenic. As regards the treatment of the changes in the eye the most important thing we can do is to assure the patient that total amaurosis does not result in the majority of cases.

169. The Limited Diagnostic Value of the Ocular Symptoms in the Other Diffuse Diseases of the Spinal Cord

Just as tabes is the only disease of the systems of fibres in the spinal cord in which the ocular symptoms possess much diagnostic value so multiple sclerosis is the only diffuse disease of the cord in which certain ocular symptoms are characteristic. We have been requested by the surgical department to see if there are any ocular changes in this patient, who is supposed to have a tumor of the spinal cord, but the changes present are very indefinite. I consider it my duty to point out the limits of the diagnostic value of the ocular symptoms in these diseases that we may be able to tell those who consult us in what diseases of the spinal cord we can contribute little to help them in the diagnosis. For this purpose we may divide the diffuse diseases of the cord into two groups: one in which the ocular symptoms have a great diagnostic value, and one in which their value is much less. Multiple sclerosis is the only representative of the first group, tumors of the spinal cord, diseases of the vertebræ, injuries of the cord, myelitis and syringomyelia belong to the second.

In the rare cases of tumor of the spinal cord we find pathological changes in the optic nerve and pareses of the ocular muscles only when cerebral complications are present, or when the tumors have extended to the brain or to the base of the skull. Disturbances in the pupil due to paresis of the sympathetic may be observed when the tumor has injured the centrum ciliospinale in the upper dorsal portion of the cord. Reflex immobility of the pupil is seldom met with.

Changes in the optic nerve, or troubles with the muscles of the eye can scarcely be produced by affections of the spinal cord caused by diseases of the vertebræ. There is only one symptom which may be noticed when the disease involves the lower cervical and first dorsal vertebræ, the paresis of the sympathetic. The ciliospinal centre is situated at this place and the sympathetic fibres leave the spinal cord in the first dorsal root. Injuries and diseases of the vertebræ at this place therefore cause the pupil to be contracted, to dilate with difficulty after the instillation of cocain, or in the dark, and the palpebral fissure to be narrowed by the sinking of the upper lid and the rising of the lower.

Likewise in direct injuries to the spinal cord in the cervical region the paresis of the sympathetic is the most noteworthy ocular symptom. In grave cases of commotio spinalis lasting pareses appear only when organic lesions have been produced, but we often observe functional disturbances after such injuries, which may be ascribed to traumatic hysteria. The visual field is concentrically contracted in such cases, there are derangements in the perception of colors, and visual troubles exist without any changes in the fundus that can be seen with the ophthalmoscope.

The diagnostic significance of the ocular symptoms is rather better in syringomyelia, which may come from many causes and in which longitudinal hollow spaces are formed, usually in the central portions of the cord, accompanied by a proliferation of the glia in the vicinity. This formation of cavities in the gray substance of the spinal cord and the medulla oblongata explains why certain eye symptoms are produced, but the diagnostic value of these symptoms is very limited. Paresis of the abducens is the most common, and shows that the disease has extended to its nucleus. The nuclei of the trochlearis and oculomotorius are almost always spared, so there is no ophthalmoplegia externa in the overwhelming majority of cases, and a lesion of the internal muscles of the eye hardly needs to be taken into account. True nystagmus is rare, while twitchings that resemble it are common. The trigeminal and facial nerves are affected with comparative frequency. Not infrequently the trigeminus is the only nerve that is impaired. The facial paresis seems to be characteristic, as it involves only one side of the face, while in progressive bulbar paralysis it is usually bilateral. But the greatest degree of diagnostic significance in this disease appertains to the paresis of the sympathetic on one side, which occurs in a fourth of the cases. The occurrence of a reflex immobility of the pupil must be ascribed to a complication with tabes, or with progressive paralysis. One point is of diagnostic value: the pathological changes in the optic nerve met with in tabes and multiple sclerosis are ordinarily absent in syringomyelia.

Another diffuse disease of the spinal cord is myelitis, in which we find at least one ocular symptom that is of diagnostic importance: optic neuritis, the characteristic symptom of which is that the vision is suddenly impaired and falls rapidly to blindness, which may last a long time and then the vision may improve. This is often the case when only an amblyopia has been produced. Another characteristic symptom is that severe pains are felt in the orbits and brows during the development of the visual disturbance. Pain is caused by the movements of the eyes, and becomes still more distinct when the attempt is made to press the eyeball back into the orbit. The course is often such that the disease of the spinal cord follows very soon after the visual disturbance. The pathological basis of the disease in the optic nerve is analogous to the changes in the spinal cord, a parenchymatous degeneration of the nerve fibres, associated with interstitial changes of an inflammatory nature. It is generally thought that the interstitial inflammation is primary, and that the degeneration and red softening of the substance of the nerve is secondary. Aside from the optic neuritis the ocular symptoms of myelitis are uncertain and not according to rule. During the amaurosis the pupils do not react, but a reflex immobility is not peculiar to this disease. Pareses of the ocular muscles are rarely met with.

170. The Ocular Symptoms and Their Signification in the Differential Diagnosis of Sinus Thrombosis

Pathological changes in the fundus are to be found in almost two thirds of the cases of otitic sinus thrombosis. First in rank comes choked disc, which is almost always bilateral. It may appear on the same side as the diseased ear, but this is unusual. The visual trouble may be very slight in spite of the great swelling of the disc, and therefore its absence must not lead us to omit the examination of the interior of the eye.

The signification of choked disc in purulent disease of the middle ear is that it almost always indicates an intracranial complication, and is therefore a valuable guide to a timely operative intervention. A papillitis may occur in an uncomplicated otitis media, according to my observation, but this certainly happens very rarely. After the operation the papillitis frequently undergoes rapid involution, but the experience of otologists shows that it occasionally increases without making the prognosis any worse.

An optic neuritis in which the circumscribed swelling of the head of the nerve is not marked is met with in otitic sinus thrombosis just as often as, and perhaps rather more frequently than this peculiar choked disc, and has the same diagnostic significance. According to the experience of otologists it indicates the presence of meningitis, abscess of the brain, or extradural abscess. We may say that choked disc and optic neuritis are to be found in 50% of the cases of this disease. More rarely the change is confined to a simple hyperæmia of the fundus. An atrophy of the nerve in consequence of sinus thrombosis is very uncommon.

Other ocular symptoms are met with, but they are of slight importance in comparison with the changes in the papilla. An exophthalmos occasionally is present, and indicates that the cavernous sinus is involved, but this condition is much more infrequent in otitic than in purely septic sinus thromboses. Pareses of the ocular muscles, especially of the oculomotorius and abducens, likewise occur when the cavernous sinus is involved. A conjugate deviation is rare. The nystagmus that is sometimes to be observed is said by *Jornsen* to be due to an affection of the labyrinth. It must be remembered that our diagnostic skill in this regard has its limits. A change in the fundus is to be found in only about one half of the cases of otitic sinus thrombosis, so the absence of these signs in any given case does not justify the conclusion that such an aural disease may thereby be excluded. The careful physician is able to recognize this condition without the aid of these symptoms.

Two other forms of sinus thrombosis, besides the otitic, are met with in practice, although they are less common, and it may be said that two thirds of all cases of this disease originate from diseases of the ear. First of these two forms is the marantic sinus thrombosis, which comes on after seriously debilitating diseases and is situated, as a rule, in the longitudinal sinus. The changes in the fundus of the eye, optic neuritis, choked disc and amaurosis, are much less common in this than in the otitic form. Troubles in the movements of the eyes are more frequent, especially the pareses of the associated muscles in the form of conjugate deviation. This symptom is due to the fact that the veins of the cerebral cortex which empty into the thrombosed longitudinal sinus are no longer able to dispose of the blood they contain, and little hemorrhages result which irritate the cortex. The spasmodic movements of the eyes, which are observed in these cases of thrombosis of the longitudinal sinus, must be looked upon as signs of such irritation. Exophthalmos is just as rare in primary marantic as it is in otitic sinus thrombosis, as is naturally to be expected, for exophthalmos can be produced in thrombosis only by an affection of the cavernous sinus.

The conditions are quite different in the septic forms of sinus thrombosis that develop after erysipelas, or after diseases of the frontal and sphenoidal sinuses. The cavernous is the sinus most apt to be affected in this form and so exophthalmos is the most common symptom, and is present in almost three fourths of all the cases. It is of practical importance to know that when the exophthalmos follows the grave cerebral symptoms the septic thrombosis has passed by way of the transverse sinus to the orbital veins, while if the orbital symptoms precede the cerebral the thrombophlebitis has extended from the orbit toward the cavernous sinus. We must notice particularly in such cases whether the thrombosis of the cavernous sinus came from the sphenoidal cavity, when the symptoms are often bilateral. It has already been shown that the optic nerve may suffer in empyema of the sphenoidal sinuses, even without sinus thrombosis. Choked disc, neuritis and atrophy of the optic nerve are also more common in this than in the marantic form of thrombosis. They are due not only to the protrusion of the eyeball, but also to the fact that nerve trunks lying in its vicinity may easily be injured by an affection of the cavernous sinus. Conjugate deviations on the other hand are much less common in septic than in marantic thrombosis of the cerebral sinuses.

XVI

Diseases of the Chorioid

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171. The Course of Examination in a Case of Chorioiditis

We find in the eye of this patient a number of roundish white spots, with more or less dense masses of black pigment at their margins, scattered over the fundus. These have been caused by a disease of the chorioid. The principal types of disease met with in the chorioid are inflammations and tumors, the latter of which is readily excluded in such a case as this.

Normal Anatomy of the Chorioid.—The chorioid is composed mainly of blood vessels, as is implied by its name, which furnish nutriment to the outer layers of the retina and conduct outward the blood from the iris, the ciliary processes, and a portion of that from the ciliary body. Its principal veins are the venæ vorticosæ, the importance of which in the change of fluid in the eye has already been discussed under glaucoma. The layers of the chorioid from before backward are: 1, the pigment epithelium; 2, the lamina elastica; 3, the choriocapillaris, or layer of capillaries; 4, the subcapillary fibrillæ; 5, the layer of vessels; 6, the lamina suprachorioidea. The first four of these layers may be taken to form a single unit of which the most important constituent is the choriocapillaris, that consists of an extremely dense system of capillaries. In the fifth layer we find small, medium sized and large vessels. The lamina suprachorioidea attaches the chorioid proper to the sclera. Chromatophore cells permeate all the vascular layers and are to be found even in the lamina suprachorioidea. They may be packed densely into the spaces between the vessels, the so called intervascular spaces, and are absent in the layer of capillaries alone.

An inflammation of the chorioid may follow an injury, or it may originate spontaneously, by way of the blood. In the latter case it will belong to one of two groups: 1, it may be an adjunct to an iritis or iridocyclitis, when it is called an iridochorioiditis or a uveitis; 2, it may be confined to the chorioid alone. The latter group may be divided for diagnostic or etiologic reasons into the nonpurulent form, also known as chorioiditis serosa et fibrinosa, and the purulent, which is also called metastatic ophthalmia. Hence, for practical purposes, we divide chorioiditis schematically into 1, Chorioiditis due to injuries; 2, Spontaneous hæmatogenous chorioiditis, which is subdivided into chorioiditis associated with iritis, iridochorioiditis, and purulent, metastatic chorioiditis, or metastatic ophthalmia; 3, Serous or fibrinous inflammation confined to the chorioid.

Inflammations of the chorioid alone are not accompanied by pain, because that tissue has no sensory nerves. The patients complain of seeing sparks and flashes, a subjective sensation we call photopsia, or of metamorphopsia, when the region of the macula is involved, which shows that a chorioiditis is not confined absolutely to that one membrane, but is very apt to affect the elements of the retina which are sensitive to light, so that straight lines look to the patient as though they were bent or kinked. Micropsia also may be present. All of these symptoms depend on a displacement or distortion of the rods and cones. Many of the patients complain of a constant flickering before their eyes.

The vision is impaired whenever the region of the macula is involved and a central scotoma is present, but otherwise the patients rarely notice the losses in the field of vision caused by the individual foci of inflammation. Such scotomata can be detected only by a very careful examination. A ring scotoma may be observed when the chorioiditis is situated chiefly in the region of the equator. This is particularly characteristic of syphilitic chorioiditis, in which the zone of disease lies chiefly in the region of the vasa vorticosa.

1. Iridochorioiditis, or Uveitis

An iridochorioiditis or uveitis is an iritis or iridocyclitis in which the involvement of the chorioid becomes a prominent feature through the presence of considerable exudates in the vitreous, and often through a marked increase of the intraocular tension. In the latter case we speak of an iritis or iridocyclitis serosa. This increase of tension in iritis serosa may be the result of an increased secretion of fluid when an inflammatory œdema is present in the ciliary body. In other cases, such as those of exclusion of the pupil, there may be an obstruction to the escape of fluid from the eye.

2. Metastatic Ophthalmia, or Chorioiditis Purulenta

In this disease pus agents, brought by the blood current, settle and multiply in the vessels of the chorioid. Its onset is characterized by signs of a severe intraocular inflammation and a marked injection of the eyeball. The foci of pus in the chorioid cause large opacities in the vitreous, so that it is very difficult to see into the eye. As the pus spreads into the retina either by extension or by metastasis we can very soon see it as a yellow mass in the vitreous. The further course of the disease depends on the kind and virulence of the microorganisms that have been implanted within the eye.

If suppuration goes on *panophthalmitis* develops. The yellow reflex from the vitreous steadily grows larger, the inflammation spreads from the chorioid and retina, synechiæ appear on the iris and a hypopyon is formed in the anterior chamber. The inflammatory œdema created by the intraocular suppuration causes the eyeball to protrude; chemosis of the bulbar conjunctiva and exophthalmos develop. If the eyeball is not exenterated its contents will break down into pus and then it will atrophy rapidly.

The clinical course is different when the metastatic ophthalmia is not so malignant. The abscess in the vitreous remains stationary, the pus agents die and the entire purulent exudate becomes gradually organized by means of granulation tissue which comes chiefly from the vessels of the chorioid. The retina is then driven forward, and a yellowish tissue, covered by vessels of the retina and traversed by others that are neoplastic, is formed in the vitreous. This is known clinically as a *pseudoglioma*. The differentiation of such a pseudoglioma from a glioma may be difficult in children. Ossification may take place within the masses of connective issue in such atrophic eyes.

Metastatic ophthalmia occurs in puerperal fever as well as after various infectious diseases, such as influenza, measles, diphtheria, pneumonia and meningitis. It may affect either one or both eyes. An ulcerative endocarditis is present in more than a third of the cases and the prognostic signification of a double metastatic ophthalmia is very serious. In puerperal fever it indicates almost with certainty a fatal result. The prognosis is not quite so bad when the metastasis in both eyes follows pneumonia or meningitis, and is not so serious in the puerperium when only one eye is affected. Unilateral metastatic ophthalmia often accompanies the milder forms of pyæmia.

3. Chorioiditis Serosa et Fibrinosa

This form is characterized by the presence of foci of inflammation scattered about in the fundus. The changes thus produced must first be studied with the ophthalmoscope, but it is only in rare cases that this examination alone is able to put us on the track of the etiology. The cause can be learned only from a general examination of the body in many cases.

We learn what form of chorioiditis is present by means of the ophthalmoscopic examination. We speak of *chorioiditis disseminata* when the foci are scattered irregularly over the entire fundus in the form of roundish or oval patches, which usually are smaller than the papilla. These patches are sometimes quite white, sometimes yellowish, sometimes reddish yellow. Little heaps of pigment are to be seen on the margins of and within the whitish spots. Frequently traces of the chorioidal vessels can be seen in them, over which the retinal vessels course, and the patches may be confluent in some places.

When the foci are arranged in groups we call the condition *chorioiditis areolaris.* When the region of the macula is the only or the principal part involved we have a *chorioiditis centralis.* This latter form is met with chiefly in myopia. It presents a large, white patch of atrophy bordered by pigment, or containing little heaps of pigment between which traces of the chorioidal vessels may be seen. When the focus is small, peculiar reticulated lines may often be seen extending out from it; these have been called cracks in the chorioid, and are considered as due to stretching in high myopia.

When the changes in the chorioid are confined chiefly to the vicinity of the papilla they are called circumpapillary. Among these may be grouped the *senile atrophy of the chorioid*, the *halo glaucomatosus*, and the *changes that take place about the papilla in myopia.*

Whatever may be the clinical designations of these forms there is a certain cycle of pathological changes in most of the varieties of chorioiditis. In the early stage the tissue of the chorioid at the focus of inflammation is permeated with lymph cells, while the retina over it is rendered cloudy at the same time. In the later stages the choriocapillaris, the pigment epithelium, and the layer of rods and cones of the retina are destroyed, while adhesions form between the two membranes.

We can determine with the ophthalmoscope whether the inflammation in the chorioid is old or not. As long as it is fresh the patches have a reddish or reddish yellow color. The infiltrations are prominent in consequence of the thickening of the chorioid, and elevate the retina a little over the diseased area. This we recognize mainly from the slightly arched course of the retinal vessels. The margins of the fresh focus of inflammation are also rather indistinct. When the inflammation has lasted a long time and has undergone involution an atrophy of the chorioid is left at the place where it formerly existed. We are obliged to draw our conclusions in very many cases from these atrophic places. The latter differ greatly in size and form; they may be roundish, oblong, pectinate, leaf shaped, and often are quite irregular. A proliferation of pigment takes place at the same time with the atrophy.

Wherever the choriocapillaris is destroyed the uniform red color that it imparts to the fundus is lost, and the large vessels of the chorioid with the intervascular spaces between them can then be recognized distinctly. Wherever in addition to the choriocapillaris the pigment epithelium has been destroyed, the fundus loses the delicate granular appearance which it causes, as well as its normal color. When the degeneration has seized upon the larger vessels of the chorioid, and when these have been rendered visible by the atrophy of the choriocapillaris and of the pigment epithelium, they look yellowish or white, and their obliteration may be recognized from the fact that they no longer contain columns of blood, or if, in spite of their thickened walls, they still contain some blood, the columns appear to be very slender. When these vessels are atrophic over large areas, we find a network of white or yellowish bands, but they may be so very atrophic as to be visible only as faint shadows and impressions. Wherever the chorioid is wholly atrophic the white sclera shines through it. When the patches are large a yellowish red, or a dirty yellow color may adjoin the white.

These pictures are complicated by the pigmentation and the involvement of the retina. An abnormal pigmentation in the form of black spots takes place wherever there is a disturbance in the circulation of the choriocapillaris and the pigment epithelium is injured. This is to be seen particularly on the margins of the atrophic places in the chorioid, where it indicates the line of demarcation between the dead and the normal pigment epithelium. *Krueckmann* has designated this black edge of demarcation as the connecting zone between the epithelium and the glia.

The retina also takes part in the pigmentation. When its outer layers suffer in a chorioiditis, a proliferation of *Mueller's* supporting cells readily takes place; processes of these glia cells of the retina then unite with the reedlike processes of the pigment epithelium, and granules of coloring matter are carried from the latter along the track thus formed. These glia cells, which have now become pigmented, proliferate, especially about the vessels. This is the reason why the abnormal pigmentation of the retina is found chiefly in this situation. Elsewhere we meet with large, round, uniformly black spots. In this case also we must imagine that a great proliferation of pigment epithelial cells takes place in consequence of the connection between the supporting tissue and the pigment epithelium. If the choriocapillaris has been destroyed in the black places the atrophy of the chorioid is covered only by the proliferation of the connective tissue and the pigmentation.

When the changes in the chorioid do not come under our observation until they are in a late stage of atrophy we must first try to differentiate whether the atrophy was brought about by a chorioiditis that formerly existed, or by some other process, for we know that degenerative diseases of the vessels can produce ophthalmoscopic pictures that are quite similar, such as are seen in arteriosclerosis and myopia, in which large portions of the capillary area are readily destroyed. A senile change that is not infrequently met with is the appearance of peculiar, colloid formations on the vitreous lamella. These are small, yellowish and lustrous, have no pigment and cause no trouble with the vision; characteristics which differentiate them from inflammations of the chorioid.

Although various etiological factors may cause or leave behind more or less similar changes in the fundus as the result of inflammation of the chorioid, yet we must try to recognize the cause directly from the ophthalmoscopic picture. This is possible in certain cases. Thus the chorioretinitis of congenital syphilis is often so characteristic that we can recognize the genesis at once. There is a diffuse atrophy of the pigment epithelium together with an alternation of minute spots of pigment with little white patches. In most cases, however, the general examination of the body is necessary to enable us to learn the probable etiology, and it must be admitted that in many cases the cause cannot be ascertained. Beyond doubt chorioiditis occurs in the course of all possible infectious diseases. The researches of *v. Michel* and of *Stock* render it probable that tuberculosis is more often the cause than was formerly supposed. Syphilis must not be forgotten. We meet with chorioiditis in women in the course of the climacteric and in abnormalities of menstruation. When the clinical examination of the body furnishes no satisfactory data we may have recourse to the aid of biological methods, such as the tuberculin test, or *Wassermann's* reaction.

The course of an inflammation of the chorioid is generally very chronic. Specific treatment may be successful, when syphilis is certainly present. In other cases we can only advise the best possible care of the eyes, prescribe potassium iodide and give subconjunctival injections of salt solution. Frequently the patients do not consult us until the chorioiditis has completely run its course.

172. Sarcoma of the Chorioid

This aged patient has noticed for some time that something was the matter with her left eye. Her vision has seemed a little cloudy at various times, separated by long intervals, but of late it has been decidedly impaired. Still her central vision is nearly half of the normal.

Although her eye appears to be perfectly normal externally, a roundish spot can be seen by means of the ophthalmoscope in the fundus near the macula, which appears in the inverted image to be three or four times as large as the papilla, and is of a grayish blue color. By the direct method it can be readily seen to rise above the level of the surrounding fundus, and we also see that the vessels of the retina make a steep ascent in order to pass over it. Such a condition suggests at once a tumor of the chorioid. Its color is somewhat different in the upright image; the yellowish red or brown surface of the growth may be seen through the retina and the peculiar network of vessels which lies upon it can be distinctly differentiated from the retinal vessels that pass over it.

A tumor of the chorioid is almost always a sarcoma. It is very seldom that any other form is met with in practice. Angiomata and gummata of the chorioid are among the greatest of rarities, and the etiology is indicated in such cases by the presence of other symptoms. It happens occasionally that a metastatic carcinoma develops in the chorioid; in these cases the primary growth is usually situated in the breast, it is found less often in the internal organs. Both eyes are affected in a comparatively large percentage of cases, sometimes one eye after the other. These metastatic carcinomas of the chorioid form characteristic flat, scalelike tumors at the posterior pole of the eye and cause severe hemorrhages. It has been observed by *Uhthoff* and by *Oeller* that a detachment of the retina caused by the development of a carcinoma may become reattached. As regards other intraocular growths sarcoma is easily differentiated from them, as a rule. Glioma is a disease of early childhood, while sarcoma is met with in the advanced years of life. The picture presented by the amaurosis of glaucoma is not the same. It is only when we meet with a solitary tubercle of the chorioid that the differentiation may be difficult; we should notice in the early stage whether any small tubercles can be seen in addition to the large one.

A sarcoma of the chorioid cannot always be recognized as clearly and distinctly with the ophthalmoscope as in this case, because it is seldom that patients come to us in the initial stages of the disease. I recall a woman who had a sarcoma of the chorioid that sprang from the ciliary body, had grown through the periphery of the iris into the anterior chamber and had caused an iridodialysis. Still her vision was good and she could not believe that she had such a malignant tumor in her eye, or consent to have the eye enucleated until the diagnosis had been confirmed by *v. Michel.*

Sarcoma of the uvea is a rare disease, one case of which is met with in about two thousand patients with diseases of the eye. It may occur at all ages of life, but is usually found in people of advanced years, between the ages of 40 and 60. It may start from the iris, the ciliary body, or the chorioid, least often from the iris, where it is most easily to be recognized, as the yellowish brownish nodules in the iris are likely to attract the attention of the patients. Sarcoma of the iris is apt to start from nævi or melanomata, and its growth is unirritating for a long time, as a rule. It becomes annoying only when it has caused the occlusion of the sinus of the anterior chamber and an increase of tension, or when the loss of vision calls attention to the fact that the growth has invaded the region of the pupil. A sarcoma of the iris may spread to the ciliary body, press the iris to one side, or extend by way of *Schlemm's* canal to the surface of the eye.

When the sarcoma starts from the ciliary body the troubles of the patient are very slight at first, because it does not cause a detachment of the retina until late in its course. This form is also to be recognized easily. If the tumor grows forward it presses between the sclera and the insertion of the iris until a dark protrusion appears in the anterior chamber. Its rupture through the iris may cause an iridodialysis. If the tumor grows rather toward the vitreous the dark protrusion can be made visible by transillumination, while by focal illumination the swelling looks yellowish, brownish, or blackish. The diagnosis is difficult only when the lens has been injured by the pressure of the tumor.

Sarcoma of the uvea starts from the chorioid in about 85% of the cases, and different stages of the disease are met with which present difficulties in the differential diagnosis. These stages are: 1, that of development without irritation; 2, that of secondary glaucoma; 3, that of the formation of extrabulbar tumors; 4, that of the formation of metastases.

During the first stage the growth of a sarcoma of the chorioid is absolutely without irritation, and the attention of the patient is drawn to it first by the visual trouble, which varies according to the site of the tumor and the injury inflicted by the latter on the retina. If the sarcoma is situated in the region of the macula a central scotoma may be present, or subjective sensations of light may occur, which are due to the stretching and irritation of the retina. A detachment of the retina must be expected to ensue, and this may come on suddenly or gradually. There are no fixed relations between the size of the tumor and that of the detachment; a very large detachment may be caused by a very small tumor. The explanation of the way in which this detachment of the retina takes place is that the tumor excites an extensive serous exudation between the retina and chorioid, but a sudden detachment may be caused by a hemorrhage from the vessels of the chorioid, or of the tumor. In many cases the onset of this detachment of the retina is the first sign we obtain that a sarcoma of the chorioid is present, and then the important question arises how to differentiate a detachment caused by an intraocular sarcoma from the ordinary form.

In the first place, the retina is so transparent in most cases of serous detachment that the red fundus can still be seen.

Secondly, the serous detachment is usually of a grayish white or greenish color, especially when the effusion contains some red coloring matter.

Thirdly, we see in a serous detachment the well known folds of the retina, and usually this membrane can be observed to float back and forth with the movements of the eye.

The form of detachment caused by sarcoma is characteristic in many cases. It rises sharply as a gibbous protrusion from its environment, exhibits no folds, the retina is tense to a certain degree, and the brown tumor, or its vessels, may still be seen even when the retina is rather opaque. If the tumor reaches far into the vitreous the details of its surface may be studied very well with the ophthalmoscope. But the effusion may be so extensive that all of these points of differentiation are more or less obliterated, and then the diagnosis becomes difficult. When the detachment is total the retina may be pressed so far forward that it is no longer possible to see into the eye. We have then two diagnostic methods at our disposal. We can test the intraocular tension, which is usually lowered in a simple detachment, while with a growing sarcoma there soon comes a time when the tension of the eye is increased, and we can employ transillumination.

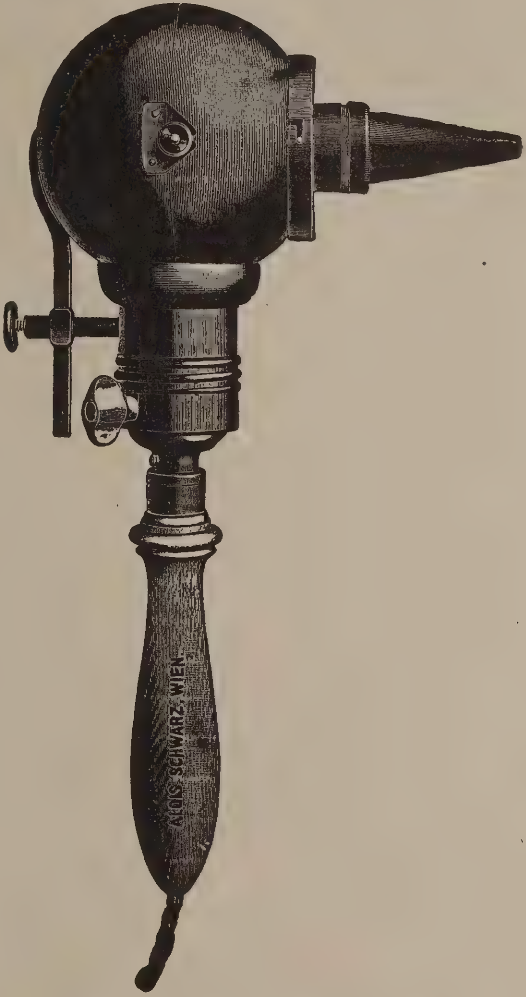


FIG. 131.—*Sach's* Transilluminator.

For the latter purpose we use the lamp devised by *Sachs*, in which the light is concentrated very strongly by being made to pass through a conically pointed glass cylinder. The eye is first cocainized and then the apex of the cone is applied to the sclera and moved about from place to place. Under normal conditions so much light passes through the capsule of the eyeball that the pupil appears red and shining. This illumination of the pupil is not impaired by an extensive serous effusion between the retina and chorioid, but if a dense tumor lies on the inner surface of the sclera the light is obstructed at this point and the pupil is darkened. This method is excellent, but naturally has its limits, for a thick layer of blood is able to suppress or decrease the light.

The only question that could be raised in a case of detachment of the retina in which the symptoms arouse the suspicion that a tumor is present is whether the detachment might not be of the chorioid, which likewise causes a dark brown, gibbous protrusion into the interior of the eye covered by the closely adherent retina. The resemblance to a tumor is great, especially when there is no floating back and forth of the retina, but a detachment of the chorioid is met with in practice only after the extraction of cataract, except as a result of grave injuries to the eye and severe hemorrhages from the chorioidal vessels. Besides, the intraocular tension is reduced when this detachment takes place, so that the differentiation is not difficult. I need hardly say that a prolapse of the iris must not be mistaken for a sarcoma of the chorioid. Finally, after the eye has been rendered blind by the total detachment of the retina and the lens has been made opaque by the nutritive disturbance in the vitreous, the diagnosis of an intraocular sarcoma is difficult and we have to rely on the question whether any symptoms have been produced that are to be ascribed to the advancing growth of the tumor.

A growing tumor must eventually cause an increase of the intraocular tension, and then the disease enters the stage of secondary glaucoma. Pain sets in and the eye becomes blind, if it is not so already. The increase of tension is caused by the growth of the tumor, the augmentation of the serous exudation within the eye and the obstruction of the outlets for the intraocular fluid. The eye assumes the appearance characteristic of glaucoma; the cornea becomes dull and loses its sensibility, the iris and lens are driven forward, the anterior chamber is made shallow, the periphery of the iris is pressed into the sinus of the anterior chamber, the pupil is dilated and is no longer capable of reaction. As long as the lens is transparent it gives the well known greenish gray reflex from the pupil. The form of the lens may be changed, or it may be pressed to one side by the growing tumor, and its posterior capsule may be ruptured.

Such a case may appear to be one of primary glaucoma, so we have to consider when the presence of an intraocular sarcoma is to be suspected and how the diagnosis is to be made. Patients with primary inflammatory glaucoma tell us, as a rule, that the vision was injured little if any up to the time of the first attack, while in sarcoma of the chorioid the vision has usually been greatly impaired or totally lost by the detachment of the retina before this onset. Again, in primary glaucoma the vision improves after the subsidence of the attack, while in sarcoma the loss of vision steadily grows worse. If the refractive media are still clear no detachment of the retina is to be seen in primary glaucoma, while it is the most striking symptom in sarcoma. Even when an accurate examination is no longer possible the reflex obtained from a retina detached by a sarcoma is grayer than that we are accustomed to see in glaucoma, and some help may be obtained from transillumination. In a case of primary glaucoma the other eye is apt to reveal a certain predisposition to the disease, while this is not true in sarcoma.

The second stage of an intraocular sarcoma may present instead of the symptoms of glaucoma, those of an iridocyclitis associated with an increase of tension. Then we find deposits on *Descemet's* membrane, a cloudy aqueous, and posterior synechiæ, which may lead to an exclusion of the pupil. The presence of a tumor may be betrayed by an ectatic condition of the sclera about the cornea, or at the equator, and the growth may finally break through the membrane. Or the course of the disease may be quite different; the eye may become phthisical after it has been blinded by a plastic iridocyclitis and cataract. In such a case the tumor may become wholly or in great part necrotic, and its growth may be suspended for a long time. We have to suppose that metabolic products are set free by the necrosis of the breaking down tumor and that these cause the iridocyclitis and atrophy. In such a case the tumor is the actuating cause of the phthisis bulbi.

Cases have been met with in which a sarcoma has developed in an eye that had long before been rendered phthisical from some other cause. Such a condition must be thought of when an atrophic eye is subject to occasional attacks of pain.

When sarcoma has been the cause of a phthisis bulbi the vision will have been completely lost at a time when a more or less useful degree would be present in a primary iridocyclitis. Phthisical eyes that contain sarcomata often have a peculiar form, to which *v. Graefe* called attention. The sagittal axis is shortened more than the diameter at the equator, so that much of the phthisical globe is to be seen in the opened palpebral fissure, and the furrows made by the recti lie more on its anterior surface. Such eyes are not as sensitive to pressure as those in which the phthisis was occasioned by a cyclitis. These are the essential symptoms by means of which the diagnosis of an intraocular sarcoma may be made during its first two stages, those in which its recognition is attended by the greatest difficulty.

When the development of the tumor has progressed until it has broken through the sclera the diagnosis is easy. If the sclera is perforated in the anterior segment of the eye dark protrusions, or black points, appear in the vicinity of the cornea. These are apt to be along the veins, for the tumor is likely to take advantage of the places of exit of the nerves and vessels. These extrabulbar nodes overlay the cornea, cover it, proliferate with extraordinary rapidity and break down into necroses and foul pus. The perforation of the tumor through the sclera at the posterior pole of the eye is not recognized so readily; in phthisical eyes particularly a long time may elapse before the exophthalmos becomes distinct. The optic nerve itself may be involved in various ways. An episcleral node may surround and infiltrate it, or the tumor may pass from the papilla into the nerve, or it may proliferate into the intermediate vaginal space. When once the tumor has reached the open orbit the condition is betrayed at once by the exophthalmos. The proliferation invades the neighboring sinuses, such as the frontal sinus and the antrum of Highmore, or passes into the cranial cavity through the superior or inferior orbital fissure, or the optic foramen, where it causes such cerebral symptoms as pareses, convulsions and loss of consciousness. The chiasm and the optic tract on the diseased side are the first to be involved. If a patient is seen for the first time when a retrobulbar tumor has already caused an exophthalmos the differentiation must be made whether the growth is a primary one of the orbit, or one coming from the interior of the eye. This is easy, because the eyeball remains intact for a great while when the tumor is one of the orbit. The differentiation is difficult only in a case of phthisis bulbi.

The fourth stage of a sarcoma of the chorioid, that of the formation of metastases, may set in quite early, or years may elapse before distinct symptoms appear. The metastases appear mainly in the liver and spread from there. They also occur in the lungs.

The prognosis is always very grave. We never know with certainty whether the tumor cells have been carried already into the internal organs or not. Metastases have been known to appear even years after an early enucleation. On the other hand some patients are permanently cured by the operation. The figures given vary, but the permanent cures may be estimated as between 25 and 40%. I am acquainted with a colleague whose eye was enucleated for this reason more than ten years ago. The earlier a sarcoma is recognized the better is the prognosis, so far as life is concerned, and we may say that the danger is over when five years have elapsed since the enucleation of the eye. This operation must not be delayed even when we have to deal with little tumors of the iris. When the tumor has perforated the sclera the orbit must be exenterated.

Some sarcomata of the chorioid consist of round cells, some of spindle cells. An alveolar construction is also met with, and we also find leucosarcomata and melanosarcomata. According to the researches of *Schieck* each tumor is at first an unpigmented round celled sarcoma and later becomes a pigmented, spindle celled, pure melanosarcoma. The finding of independent unpigmented sarcomata in the chorioid has been confirmed by others. The greater part of the pigment in a melanosarcoma is in the pigment cells, but another variety may be found to which *Vossius* and others have ascribed a hæmatogenous origin. The starting point of a sarcoma of the chorioid is in the middle and deeper layers of the vessels, but the true nature of its development is still unknown, as is the case with many other tumors. Sometimes traumatism may be the exciting factor, but we must be very careful in passing judgment to this effect,

XVII

Diseases of the Optic Nerve

173. Neuritis Optica and Its Etiology

This middle aged man complains that he has not been able to see well for some time past, and we find on examination a serious lesion in the head of the optic nerve in each eye. The papilla of his right eye is much reddened, its margin is so indistinct that it cannot be distinguished from the rest of the fundus, and its exact boundaries cannot be defined. A gray, striated opacity extends from the surface of the papilla quite a distance into the retina. Another characteristic of optic neuritis is to be found in the vessels; the arteries seem to be normal, but the veins of the retina are engorged and tortuous. Little streaks of blood can be seen lying near the vessels in some places.

In the other eye we find the disease in a more advanced stage. Signs of atrophy can be detected after an optic neuritis has lasted for some time, and this neuritic atrophy is to be differentiated from the simple, genuine atrophy of the optic nerve chiefly by the facts that the papilla looks gray rather than white, that its substance is cloudy and its margins indistinct, and that the veins are still tortuous, although the arteries frequently appear as white lines.

No conclusions as to the degree of vision present can be drawn from the ophthalmoscopic picture alone. The whitish gray color that follows an optic neuritis need not be caused by a total atrophy of the optic nerve fibres; the discoloration may be due to an abnormal proliferation of connective tissue. The test of the vision, of the visual field and of the color sense alone can give us positive information in regard to this matter. This is true of quite a number of diseases of the fundus. No matter whether such a case is seen at the beginning, as a fresh optic neuritis, or later, when the neuritis has resulted in atrophy, the ophthalmoscopic examination solves only one problem: the ophthalmoscopic diagnosis of an optic neuritis, or of a neuritic atrophy, is only one step toward the clearing up of the etiology of the disease from which the patient is suffering, and this it is our duty to investigate. In order to do so we must be certain of the fact that we have to deal with an inflammation of the optic nerve and then we must learn its cause by means of a differential diagnosis.

I. Anatomical Comparison between a Normal and an Inflamed Optic Nerve

I have to proceed on the supposition that the ophthalmoscopic picture presented by the normal optic nerve is well known. There is only one preliminary question to be settled when we see an optic nerve that presents an appearance which excites our suspicion of a neuritis. This is whether it may not be a so called *pseudoneuritis.* The margins of the papilla may appear to be indistinct and the head of the optic nerve may look reddened in hypermetropia and astigmatism. If at the same time the veins are rather tortuous a neuritis is suggested at once, but there is no opacity of the tissue of the papilla and the correct diagnosis can be made by a careful test of the functions of the eye and by observation. When one is in actual doubt as to the presence of an inflammation in the optic nerve he should look into a normal eye with the ophthalmoscope in order to make a comparison.

Anatomy of the Normal Optic Nerve

The optic nerve is composed of a trunk and three sheaths, which are direct prolongations of the three membranes of the brain and are accordingly known as the dural, the arachnoidal and the pial sheaths. The firm, tendinous dural sheath joins the optic nerve before its passage through the foramen opticum, in which it forms the periosteum, and loosely surrounds the nerve from that point on. The arachnoidal, a delicate membrane, lies on the inner surface of the dural sheath, while the pial sheath is closely attached to the nerve. The space between the dural and pial sheaths is known as the intervaginal space, and is a lymph cavity which communicates with the subdural space of the brain. It ends in a cul de sac at the sclera and may be divided into a subdural and a subarachnoid space which are imperfectly separated by the arachnoid sheath. It contains septa that arise from the pial sheath, consist of connective tissue with few elongated cell nuclei, and contain vessels—both the central vessels of the optic nerve, which supply the retina, and smaller ones which supply the nerve itself. Within the septa lie the bundles of nerve fibres with the neuroglia. The individual nerve bundles consist in turn of fine nerve fibres, axis cylinders surrounded by delicate sheaths. It may be seen in longitudinal sections stained by *Weigert* that the medullary sheaths of the optic nerve fibres, which are stained black, end at the lamina cribrosa. Within the eye the optic nerve fibres have no medulla under normal conditions. When the fibres lose the sheath of *Schwann* the enveloping delicate neuroglia follows the medulla. Some of the nerve fibres are thinner, some thicker; the thinner ones are those employed in vision, the thicker ones appertain to the pupil.

After passing through the lamina cribrosa these fibres spread out in the form of a funnel, the bright centre of which in the optic disc corresponds to the so called physiological excavation. The reason this appears to be so bright is because the whitish, lustrous connective tissue of the lamina cribrosa is permitted to shine through distinctly in consequence of the spreading out of the fibres. It can readily be imagined that this would be more indistinct if the papilla were inflamed.

The optic nerve is supplied in the orbit by the vessels of the pial sheath, but its principal vessels are the arteria and vena centralis. The former enters the optic nerve about 15 mm behind the eyeball, coming from the ophthalmic artery. Anastomoses exist within the lamina cribrosa between the posterior short ciliary arteries and the central vessels to form *Zinn's* or *Haller's* circular arterial plexus, from which arise the independent cilioretinal vessels in the papilla that furnish nutrition to certain areas of the retina. The central vein leaves the trunk of the optic nerve rather nearer the eyeball than the entrance of the central artery, usually in the lower outer quadrant. It opens ordinarily into the ophthalmic vein, sometimes directly into the cavernous sinus.

Anatomy of the Inflamed Optic Nerve

The papilla is swollen. Along the small blood vessels are infiltrations of cells which render them particularly distinct. The substance of the papilla is infiltrated with numerous nuclei and vessels, there is an œdema between the septa, and the fibres appear to be more loosely connected. The œdema and the great plethora of nuclei and vessels in the layer of fibres is particularly noticeable, and extensions of these may be seen in the granular layers of the retina. At first the nerve fibres are normal, but later are destroyed by the products of inflammation. The medullary sheaths are lost, the axis cylinders are destroyed. Hand in hand there is a proliferation of the glia, a multiplication of the cells of which can be seen in the cross sections.

This anatomical comparison enables us to understand the clinical picture presented by an optic neuritis. The hyperæmia in the optic nerve, the great fullness of its minute and very numerous vessels, causes the abnormal redness of the papilla. This appears ophthalmoscopically in a marked case of neuritis as a dusky, partly livid tone of color. The small vessels cannot be perceived with the ophthalmoscope. This peculiar tone of color is not present in a pseudoneuritis. In true neuritis a cloudiness of the tissue in the optic nerve and in the adjacent parts of the retina is added to the abnormal redness. This cloudiness is due partly to the hyperæmia, partly to the serous infiltration. The individual layers, particularly that of the nerve fibres in the retina, are loosened up, and in addition there is a more or less marked swelling of the papilla. These factors, the hyperæmia, cloudiness and swelling of the tissue, cause the margin of the head of the optic nerve to become indistinct, the lamina cribrosa to become invisible, and the deeper lying portions of the retinal vessels to look as though they were behind a veil. The degree and extent of the inflammatory changes vary a great deal. When the infiltration and serous saturation is widespread the swelling extends into the surrounding retina and we

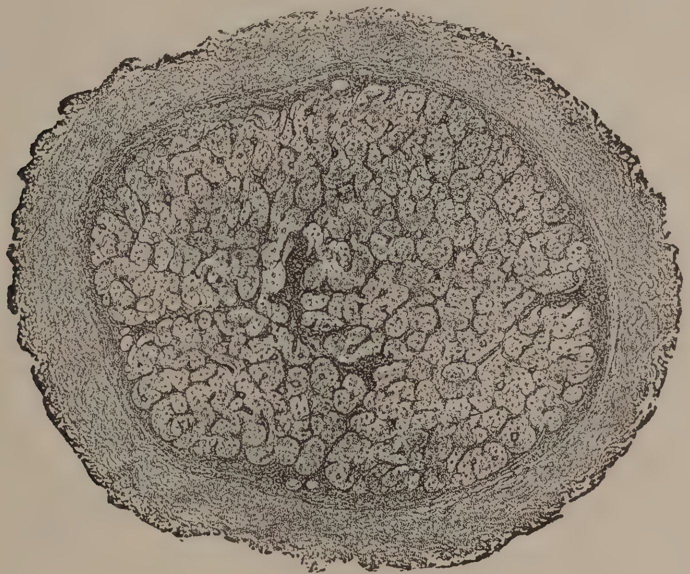


FIG. 132.—Neuritis Optica.

speak of a neuroretinitis, but when the inflammation is ophthalmoscopically confined to the place of entrance of the optic nerve it is called a neuritis.

Although the degree of swelling may vary considerably we make a sharp distinction between an optic neuritis and a true choked disc. The anatomical absence of signs of inflammation and the peculiar projection of the optic nerve like a mushroom justify the distinction between the two.

II. Etiology of Neuritis Optica

We must bear in mind that the optic nerve is nothing else than a protruded portion of the white substance of the brain, just as the retina may be considered one of the nervous centres. The neuroglia of the brain can be traced far into the optic nerve. This explains why inflammations of this nerve usually develop from an inflammation of the surrounding membranes, the meninges. Most inflammations start in the connective tissue septa and thence involve the trunk. An inflammation of the meninges of the brain may very readily extend to their immediate connections, the sheaths of the optic nerve, and so optic neuritis is a very common symptom of meningitis. As the causes of such a cerebral disease may vary a great deal, it is evident that quite a number of morbid processes may excite an inflammation of the optic nerve. Whatever may be the cause of an optic neuritis its ophthalmoscopic picture remains the same and does not always reveal its etiology, because the optic nerve must react more or less with the same anatomical changes to the greatest variety of infectious diseases and toxines. Just as we always meet with posterior synechiæ in an iritis, whatever its etiology may be, so we meet with redness, swelling and obscuration of the margins of the disc in an optic neuritis, whatever may be its etiology.

An investigation of the vicinity of the eye and of the entire organism is necessary in order to discover the real cause of an optic neuritis, so we need first to inquire in what diseases the optic nerve is involved in the form of an optic neuritis in order that we may be in a position to decide what fundamental disease is present in any particular case. I have arranged these diseases systematically in the following groups: Group 1, optic neuritis caused by diseases in the vicinity of the eye; *a*, diseases of the orbit; *b*, diseases of the accessory sinuses. Group 2, optic neuritis from diseases of the brain and abnormalities of the skull. Group 3, optic neuritis from diseases of metabolism. Group 4, optic neuritis from poisoning. Group 5, optic neuritis from infectious diseases.

Group 1. Neuritis Optica Due to Diseases of the Orbit and of the Accessory Sinuses

Any disease of the orbit, like a cellulitis or a periostitis, which involves the vicinity of the optic foramen will cause an optic neuritis of one eye, and the vision may be lost before the descending neuritis has become visible on the papilla. An optic neuritis may also be caused by an erysipelas in the vicinity of the eye and may result in atrophy and blindness.

The question whether a disease of the accessory sinuses is present, especially of the posterior ethmoidal cells or of the sphenoidal sinus, is very important. The optic neuritis caused in this way may affect either one or both eyes. Frequently the trouble begins with an acute disturbance of vision, usually a central scotoma, after which the neuritis appears. When we can find no plausible and satisfactory explanation of an optic neuritis the accessory sinuses must be carefully examined, for in many cases immediate operative treatment can save a part of the vision.

Group 2. Neuritis Optica Due to Diseases of the Brain and to Abnormalities of the Skull

The etiology is easily recognized when abnormalities of the skull are present. Optic neuritis is met with in scaphocephalus and in oxycephalus, especially in the form of the latter known as peaked skull (Turmschaedel), among which we find cases of distinct neuritis associated with atrophy. *v. Michel* found in such a case a considerable constriction of the optic foramen, which caused the neuritis by engorgement of the intervaginal fluid and compression of the optic nerve.

Optic neuritis due to diseases of the brain is much more important. An involvement of the optic nerve is a very frequent and an extremely valuable diagnostic symptom in meningitis. Another disease of the central nervous system in which optic neuritis may be a valuable early symptom is multiple sclerosis, and this disease must be borne in mind when no other reason for its appearance can be detected. In many of these cases the neuritis affects one eye only and the visual disturbance may at first take the form of a central scotoma. An early and accurate neurological examination is necessary in a case like this, and such an examination should be made in all cases in which the cause cannot otherwise be detected.

Group 3. Neuritis Optica Due to Diseases of Metabolism

In this connection I need only mention the occurrence of neuroretinitis albuminurica and diabetica. Optic neuritis is not very uncommon in women during lactation and menstrual disturbances. The true nature of these troubles has not yet been investigated. It is not yet known whether the neuritis met with in the course of lactation is due to an autointoxication with the toxic products of metabolism, or whether the weakness of the general constitution furnishes a sufficient explanation. Intercurrent infections at these times are also to be taken into account. Fortunately this neuritis usually leads only to a partial atrophy of the optic nerve, so that useful vision is preserved. The details of the connections between disturbances of menstruation and optic neuritis are equally unknown, and yet sometimes we are obliged to assume such a connection to exist.

Those cases in which we meet with an affection of the optic nerve after great losses of blood, e.g., after hemorrhages from the stomach, belong more or less in this category. Some days after such a hemorrhage the papilla appears pale and cloudy with its margins obscured by an opacity that extends into the retina. The ophthalmoscopic appearance of the papilla differs from that of a grave optic neuritis in the condition of the vessels, which look smaller and paler than normal. The visual disturbances appear a few days after the loss of blood. The final results vary. Ordinarily the ophthalmoscopic picture of atrophy of the optic nerve persists, but still a good part of the vision may be preserved.

Group 4. Neuritis Optica Due to Poisons

The amblyopia caused by alcohol and tobacco needs only to be mentioned. A marked optic neuritis may occur in chronic lead poisoning. Quinine is one of the drugs that may produce a high degree of amblyopia, but the ophthalmoscopic picture differs from that of a neuritis in that the papilla is strikingly pale and the vessels very small. The field of vision is contracted concentrically and a central scotoma is rarely present. Usually the visual disturbance is transient. Pathological changes have been found in the ganglion cells of the retina after experimental poisonings by *Ward Holden*, and by *Birch-Hirschfeld.*

Group 5. Neuritis Optica Due to Infectious Diseases

There is scarcely an infectious disease in the course of which an optic neuritis is not occasionally observed. It is met with in *measles, scarlet fever, diphtheria, typhoid fever, influenza, whooping cough, pneumonia, acute articular rheumatism,* and most of all in *syphilis.* It happens not so very seldom that a quite acute and very serious visual disturbance appears in one or both eyes, the cause of which can be recognized at the time, or a little later, to be an optic neuritis or a neuritic atrophy, after catching cold, or after a thorough drenching of the body. The basis of this trouble is rheumatic, but we are still completely in the dark as to the way in which it is brought about. In one such case I was able to find a disease of the sphenoidal sinus as the connecting link, but in other similar cases the accessory sinuses were healthy.

Above all we must always determine whether the optic neuritis is not of *syphilitic origin.* Careful examination often reveals very characteristic thickenings of the walls of the retinal vessels with white striæ, but in many cases *Wassermann's* reaction alone is able to guide us to a correct diagnosis.

In certain very rare cases the optic neuritis is due to a *hereditary taint,* the causes of which are little known as yet. This disease usually appears quite suddenly in the members of the affected families as a central scotoma. Both eyes may be affected at the same time, or one may be attacked some time before the other. Its onset takes place about the age of puberty, and more than 80% of the male members of such families suffer. Ophthalmoscopically there is at first a hyperæmia and slight cloudiness of the papilla which gradually grows pale, and, as a rule, the temporal half is paler than the nasal, a condition that corresponds to the nature of the visual disturbance. The peripheral vision is usually more or less preserved and in the cases that I have thus far seen th patients are able to see enough to go about alone.

The treatment of optic neuritis is naturally to be directed toward its etiology. If the cause lies in the orbit, or in the accessory sinuses, these diseases must be treated. In other cases sweating, preparations of salicylic acid, potassium iodide and antisyphilitic treatment are to be employed.

174. Simple Atrophy of the Optic Nerve and Its Causes

Ophthalmoscopic Picture of Genuine Simple Atrophy of the Optic Nerve

The cardinal ophthalmoscopic signs of simple atrophy of the optic nerve are: First, the papilla appears in the inverted image as white and shining, greenish white, or bluish white, with the gray stippling of the lamina cribrosa distinctly visible, but to make the diagnosis certain the nerve must be examined in the upright image, for the disc frequently appears fairly white in the inverted image when it can be seen to have a reddish color in the upright.

Second, the margins of the optic disc are sharply defined in genuine atrophy, a point in which it differs from neuritic atrophy.

Third, we find an atrophic excavation when the atrophy is complete. This excavation is total and extends to the margins, but it is shallow, so that the vessels do not appear to be sharply bent where they pass from the nerve into the retina, they pass over the margin of the papilla in a gentle arch.

Microscopically the entire nerve appears to be attenuated by the atrophy, and when we have an opportunity to see it in a fresh condition it looks gray and glassy. In the cross sections the trabeculæ of connective tissue appear to be thickened and their nuclei to be increased. The nerve fibres are absent in the retiform spaces of the septa and in place of them we find a delicately fibrous tissue; the nerve fibres have degenerated into fine nonmedullated fibrils. The mass frequently contains drops of myelin. In a case of tabetic atrophy the process starts in the layers of ganglion cells and of nerve fibres of the retina and mounts upward along the nerve. When the changes that are demonstrable anatomically and visible clinically are taken together it is evident that a lowering of the central vision, a contraction of the visual field, an impairment of the color sense and, finally, amaurosis must be the result.

The course of a simple progressive atrophy of the optic nerve is marked, as a rule, by a gradual deterioration of vision. If the visual field is tested in the beginning of the disease the first thing that can be noticed is a contraction of the field of color vision, and that the limits for green in particular are contracted. Then the field for white gradually grows smaller and the central vision fails, until at last it sinks into amaurosis. Years often elapse before this final stage is reached.

Various causes operate to produce simple progressive atrophy of the optic nerve and we cannot differentiate them by means of the ophthalmoscope alone. It is one of the symptoms produced by certain diseases of the nerve itself. Tabes is the cause in at least 40% of all such cases, and as almost every alternate case of optic atrophy is a symptom of tabes we must investigate for the presence of one or more of the other marked symptoms of this disease as soon as we are certain that the nerve is atrophic. Such symptoms are reflex immobility of the pupils, absence of the patellar reflex, pareses of the muscles of the eye, motor or sensory disturbances, gastric crises, a sense of constriction about the body, lancinating pains in the limbs, and paræsthesias.

Next we have to think of progressive paralysis, as atrophy of the optic nerve occurs in about 5 to 8% of the cases of this disease.

Affections of the optic nerve are still more frequent in multiple sclerosis, in which, aside from the optic neuritis, the atrophy of the nerve is usually incomplete. The papilla is only partially atrophic and frequently only one eye is affected. The disease is certainly to be considered neuritic, but the neuritis in the trunk of the optic nerve may run a brief course and leave behind only the picture of a simple atrophic discoloration of the papilla.

A tumor of the brain may cause a genuine optic atrophy when it compresses the optic nerve and interrupts its conduction. This is particularly the case in acromegaly, in which the atrophy is due to the compression of the chiasm and of the optic nerve by the tumor of the hypophysis. When we see an atrophy of only one optic nerve we must also think of a fracture of the base of the skull in which the optic nerve has been torn or compressed in the optic foramen.

Occlusion of the central artery may also give rise to the picture of optic atrophy, but the margins of the papilla are not sharply defined and the extreme smallness of the vessels indicates the cause in such a case. A simple optic atrophy may also appear in old age as the result of arteriosclerosis. In a general way we may make this comparison between the optic atrophy of tabes and the descending atrophy of other diseases: in tabes the signs of atrophy are to be seen at the time the visual trouble begins; in the descending atrophies the visual trouble may be present before we can positively recognize the paleness of the optic disc with the ophthalmoscope. There are quite a number of etiological factors to be remembered when we see a case of genuine optic atrophy, and, even though we ourselves are therapeutically impotent, we can in very many cases recognize the onset of diseases early enough to enable them to be at least arrested or ameliorated by proper treatment.

175. Disease of the Papillomacular Bundle of Optic Nerve Fibres and the Etiology of Retrobulbar Neuritis

The history of this patient is as follows: T. R., laborer, 37 years old. Father killed in an accident; mother, brothers and sisters well. No family history of nervous or mental disease or of convulsions. Development normal. Had typhoid fever when 9 years old, otherwise has never been sick; no venereal disease. Drinks every day, is drunk for three or four days every four weeks, but works regularly. He has complained that he could not see well for a year past. About three months ago he began to have severe headaches and two months ago he had an attack of convulsions, on account of which he was taken to the hospital. On the day of his discharge from the hospital be became restless and fearful, saw cats, rats, mice, and a great number of men dressed like clergymen who approached him without speaking. He picked threads from his clothing. In short he had an attack of the transient delirium of chronic alcoholism, which improved quickly in the neurological department. The following is a brief abstract of the neurologist's report:

Large, strong, well nourished man. Color of face good. Facialis and trigeminus intact. Tongue is protruded straight forward, trembles slightly. Patellar reflex +, symmetrical; Achilles reflex +; ankle clonus on each side, more marked on the left than on the right. Abdominal reflex rather weak on each side; cremasteric reflex +; arm reflex +. No *Babinski*, no *Oppenheim*. Gross strength not reduced. Sense of position distinctly impaired; touches are not always announced promptly; slight reduction of sensitiveness to pain in the limbs; sharp and blunt are well distinguished, though the answers are not always certain; pricks with a needle perceived everywhere; heat and cold are well distinguished. When the eyes are closed he wavers somewhat and he stands insecurely on each foot. No distinct ataxia in walking. In the heel knee test he is not quite certain of touching the patella; distinct atactic motions on drawing a triangle or a circle in the air. Able to carry a glass filled to the brim with water to his lips without spilling. Peripheral arteries rather tortuous and rather rigid. Pulse regular, strong, 72 beats per minute. Tremor of the outstretched hand which is not increased by grasping an object. *Quinquaud* +. Thyreoid not enlarged. Psychically, patient makes a rather dull, apathetic impression. Localization as to time slightly impaired, as to place better. Feels sick, but cannot tell where. Is unable to say words backward, begins rightly, but then always says them forward. Often makes omissions and mistakes in his answers. Questions requiring judgment very badly answered. Calculation very bad. Memory for figures greatly impaired, a number of four figures forgotten in one minute. Examination of the blood for syphilis negative.

It is not without reason that I have given this clinical history, for in this patient the eye symptoms are of predominant interest.

I. The Ophthalmoscopic Picture and the Condition of the Field of Vision in Retrobulbar Neuritis

The temporal half of each papilla is white, while the nasal portions retain their normal color. The temporal half of the normal disc is rather brighter than the other, but the difference is so marked in this case that the atrophic temporal portion of the papilla seems to form a sector distinct from the rest. This is due to a sharply circumscribed partial atrophy of the optic nerve in the temporal half of the papilla, which gives rise to the characteristic picture of a retrobulbar neuritis confined to a certain bundle of fibres, the papillomacular. The changes to be seen with the ophthalmoscope are not always as well marked as they are in this patient, especially not at the beginning of the disease. The condition may be absolutely negative at first, or the papilla may be a little reddened and the pallor on the temporal side may appear later. For this reason we have to depend at the commencement of the disease on the subjective statements of the patient and the test of the fields of vision.

The most important symptom mentioned by the patient is that he has nyctalopia, that he is able to see better in the evening than during the day. A partial explanation of this is that the pupil dilates as the amount of light is reduced, so that the peripheral portions of the retina take a greater part in the act of vision, but even when the vision is not really better the annoying sensation of dazzling passes off and the patient feels that he can see better.

A central scotoma, which is usually negative, can be demonstrated early in disease of the papillomacular bundle of nerve fibres. Red and green colors appear to be darker than they should be, and color perception is finally lost in this central area. If the development of the scotoma is followed carefully we find that a little central defect for red and green appears first, an enlargement of the blind spot follows, and later the two blend, so that the scotoma has an oval form corresponding to the papillomacular area of the retina. Colored objects appear at first to be duller, then to be colorless; finally they are no longer to be seen and the perception of white is lost. The scotoma is then absolute. The outer boundaries of the fields for white and for colors may be normal, or they may be contracted to varying extents according to the degree of atrophy, for it must not be imagined that this papillomacular bundle has sharply defined limits, and the disease may involve more or less the other fibres of the optic nerve.

In order to demonstrate a central scotoma for red and green the examiner may place a little colored object on the tip of his nose and cover it with his finger; the patient is then told to close one eye and fix the other on the tip of the examiner's nose; then, when the examiner removes his finger the patient is unable to name the color if a central scotoma is present. While he continues to fix his eye on the same point the little colored object may be moved in a circle before his eye and the color is recognized at once. The central perception of color can be ascertained in this very simple way, and then the scotoma may be measured accurately on the perimeter.

II. Elucidation and Pathology of the Disease

The reason why such a sharply defined partial atrophy occurs and produces such a typical clinical picture is because a number of diseases affect in a selective manner only a certain bundle of fibres in the trunk of the optic nerve, the so called papillomacular bundle, the degeneration of which can be demonstrated microscopically. The position of this bundle cannot as yet be shown in the normal optic nerve by means of an elective stain, but the diseases that affect it have revealed its position, which varies in different parts of the nerve. Near the foramen opticum and in the upper part of the orbital portion of the nerve it lies in the middle of the trunk about the central vessels and is therefore known as the axial bundle, but it lies to the temporal side of the vessels at the place where they enter the eye. This is because the optic nerve rotates somewhat about its axis during its course through the orbit; in this part of the nerve the bundle forms a wedge shaped sector with its apex at the central vessels, and it occupies the temporal side of the papilla when the nerve enters the eye. This is the reason why the temporal half of the papilla becomes pale when the papillomacular bundle undergoes degeneration. These fibres are of great importance, because they supply the macula and provide for the central vision. They follow a curvilinear course from the temporal margin of the optic nerve to the macula, and when they have lost their functional power a central scotoma must result. In this manner are explained the two most important symptoms of disease of the papillomacular bundle, the central scotoma and the partial, temporal atrophy of the optic nerve.

The degeneration of the optic nerve fibres is only the final stage of the pathological process. The researches of *Vossius*, *Samelsohn*, *Uhthoff*, *Bunge*, and *Schieck* have shown that it is preceded by an optic neuritis which may run downward from the optic foramen to the eyeball, or may start near the eyeball and extend upward. The interstitial tissue proliferates, is rich in nuclei and vessels, atrophies, and so may interfere with the functions of the fibres. These changes in the interstitial tissue are primary in many cases, while the disease of the parenchyma is a secondary symptom. The primary cause may also be a sclerosis of the vessels. I believe, however, with *Birch-Hirschfeld*, that in many cases we cannot accept the theory of an interstitial neuritis, but must assume a quite specific toxic action upon the ganglion cells and axis cylinders of the fibres of the optic nerve. We can recognize this disease with the ophthalmoscope only after the first changes can be perceived on the temporal half of the papilla. Sometimes these changes consist of a suspicious hyperæmia of this portion of the disc, but we can be certain only after signs of atrophy have appeared.

III. Etiology of Retrobulbar Neuritis

Two groups of diseases are capable of producing disease of the papillomacular bundle of fibres and thereby a central scotoma: chronic intoxications and certain general diseases.

a. Retrobulbar Neuritis in Chronic Intoxications

Alcohol and tobacco amblyopia calls first for consideration. Either alcohol or tobacco alone may cause a retrobulbar neuritis, but usually both of these poisons work together to produce it. Methyl alcohol can produce an acute amblyopia and even amaurosis. The cheap kinds of tobacco and alcoholic liquors are the most dangerous. The patients are, as a rule, middle aged and elderly men, who complain of a progressive impairment of their vision. When the temporal half of the papilla is already atrophic and the scotoma is absolute, no restitutio ad integrum can be obtained, but when the disease has not progressed so far the interstitial neuritis of the optic nerve, due to the abuse of alcohol and tobacco, has a comparatively good prognosis. Timely treatment and absolute abstinence may improve the central vision and even restore it to normal, provided that the patient does not yield to temptation. Aside from absolute abstinence the treatment consists of sweatings, potassium iodide, carefully dosed injections of strychnine and protection of the eyes.

In *chronic lead poisoning* the onset of the visual disturbance may be quite acute, but usually other symptoms of plumbism are present. Frequently a neuritis can be detected with the ophthalmoscope, or it may be that only a central scotoma may be made out at first.

A central scotoma, usually without involvement of the peripheral parts of the field, is met with in the *carbon bisulphide poisoning* of workmen employed in the manufacture of rubber goods. The same form of amblyopia may be produced by such substances as salicylic acid, carbolic acid, iodoform, morphin, antipyrin, nitrobenzol, as well as by atoxyl, which has proved of late to be such an efficient remedy in the treatment of sleeping sickness.

b. Retrobulbar Neuritis in General Diseases

First among the general diseases is *multiple sclerosis.* The foci of degeneration in this disease seek out, with a certain predilection, the papillomacular bundle in the optic nerve, and whenever we meet with a central scotoma of one or both eyes we must be on the lookout for other symptoms of multiple sclerosis.

Thus far I have spoken only of chronic disease of the papillomacular bundle, but we occasionally, though not very often, meet with a neuritis which is quite acute in its onset and in which this bundle of optic nerve fibres is particularly involved. Such patients become totally blind in a few days or hours. Usually they can still perceive objects peripherally, but not those that are directly in front of them. It is becoming more and more evident from clinical experience that multiple sclerosis is the cause in most of these acute cases, yet the etiology of some of them has not yet been explained. Even when the amaurosis has lasted for days the peripheral vision may improve, although the central disturbance remains.

A good diagnostician must also know that a central scotoma is not uncommon in *diabetes.* Usually the patients are elderly and very much broken down, or at least in a late stage of the disease. Diabetes may also cause an acute blindness through retrobulbar neuritis, but we rarely find marked signs of inflammation on the papilla.

Finally, *diseases of the accessory sinuses of the nose* may cause retrobulbar neuritis, and some cases have to be ascribed to febrile general diseases and to catching cold.

176. Choked Disc and Tumor of the Brain

I. Ophthalmoscopic Picture of Choked Disc and the Behavior of the Vision

The head of the optic nerve protrudes like a mushroom into the vitreous of both eyes of this patient. The papilla is greatly swollen and is of a gray red color. Its tissue has a peculiar glassy cloudiness which extends a little into the surrounding retina and obscures its margins. In the upright image a gray striation can be seen on the papilla, which corresponds to the course of the swollen and œdematous nerve fibres. The fourth principal symptom is the behavior of the vessels. The physiological excavation disappears in consequence of the swelling of the funnel that contains the vessels. This swelling affords the means by which the great prominence of the papilla can be recognized. The arteries appear to be small, while the veins are very large, dark red and very tortuous. Entire portions of the veins are hidden in the cloudy tissue in some places and come to the surface in others, so that they seem to be interrupted in their courses, although they are in fact only covered by the swollen and opaque tissue. At the places where the vessels are to pass into the retina they can be seen to descend the slopes of the prominent papilla in strong curves. Little hemorrhages may be observed near the vessels between the striæ of the tissue of the disc. In some cases we find in addition to the hemorrhages white striæ or plaques formed mainly by nerve fibres that have undergone varicose degeneration. Even a picture similar to that of retinitis albuminurica, with the stellate figure at the macula, may sometimes be noticed in choked disc.

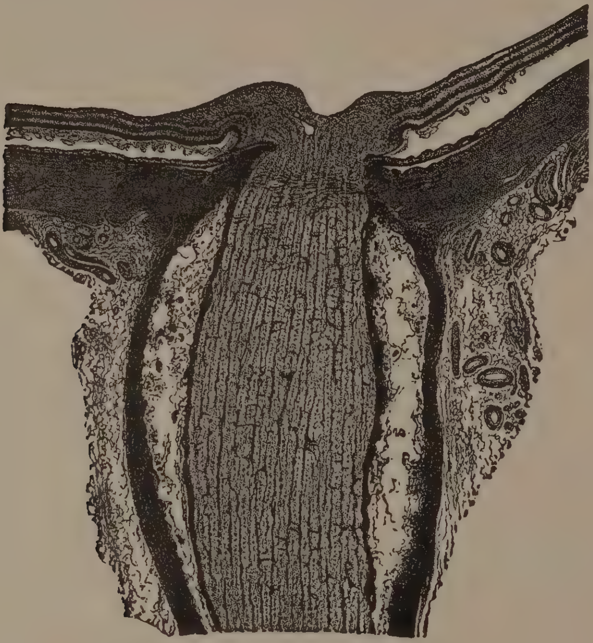


FIG. 133.—Choked Disc.

Next we must measure accurately the degree of swelling of the papilla. While we are making an examination by the indirect method it will be seen that if we move the lens up and down the anterior of two points lying near each other on different levels will pass over the posterior. This phenomenon is particularly marked when the choked disc is very prominent, but a more exact measurement can be made by the direct method. The refraction of the surface of the papilla is first measured with the ophthalmoscope and then that of the retina. A difference in level of 1 mm corresponds to a difference of refraction of 3 dioptres. If the refraction of the retina is emmetropic and that of the top of the papilla is +3.0 D, the height of the choked disc is 1 mm. If the patient is hypermetropic the hypermetropia of the disc is still greater, if he is slightly myopic the papilla may be emmetropic or hypermetropic, and in high myopia the myopia of the papilla is less than that of the retina.

Another point that we have to decide ophthalmoscopically is whether the choked disc is fresh or old. When the condition has lasted some time, or is undergoing involution, atrophic changes can be seen. The prominence of the papilla decreases, but the veins remain tortuous and its margins are not as distinct. Its color becomes brighter and finally blue gray, but the lamina cribrosa remains invisible.

The vision must be determined before we can proceed in our investigation and reach diagnostic conclusions. In this patient it is still nearly normal, a fact that is very instructive. Grave changes may take place in the papilla without betraying themselves in disturbances of the vision. Hence the ophthalmoscope must be used in all cases in which there is any reason to suspect the presence of a choked disc.

II. Pathology of Choked Disc

The mushroomlike swelling of the head of the optic nerve is due primarily to œdema. The entire substance of the nerve is softened. We also find proliferation of the nuclei and distended capillaries, which are in part new formations. In the later stages we meet with signs of inflammation, cellular infiltrations, hemorrhages, thickenings of the nerve fibres and finally destruction of the latter, proliferation of the connective tissue, increase of the nuclei, and inflammatory changes in the sheaths of the vessels. In the final stage the changes correspond to the signs of neuritic atrophy.

Aside from the proliferations of the endothelium the marked ampullalike dilatation of the intervaginal space is very striking. It is plain that an engorgement of lymph must have occurred at this place. This hydrops of the intervaginal space is absent, or at least less distinctly marked, in a good many cases.

III. Etiology of Choked Disc

The great diagnostic importance of choked disc depends on the fact that it calls attention to the presence of a cerebral disease, usually a *tumor of the brain.* Ninety per cent. of all intracranial tumors are associated with a choked disc. The nature of the tumor is not indicated; it may be of the bones, membranes, or substance of the brain; it may be a sarcoma, a glioma, a carcinoma, a gumma, a tubercle, an aneurysm, or a cysticercus. Neither is the choked disc a guide to the size of the tumor, for it may be of high degree when caused by a very small growth, as, for example, when a small tumor is situated in the cerebellum.

Double choked disc is met with not only with tumors of the brain, but also with *abscess of the brain, chronic meningitis of the base,* especially when of syphilitic origin, *hydrocephalus* and *certain congenital* deformities of the skull. It may occur in one or both eyes in cases of gumma of the base of the skull, in *sinus thrombosis,* and in *hemorrhage at the base of the skull.* Choked disc of one eye alone is usually met with in connection with inflammations due to *neoplasms in the orbit.* A double choked disc occurs in rare cases of such general diseases as *nephritis, lead poisoning, anæmia, multiple sclerosis,* and a number of acute infectious diseases, but, as a rule, the picture presented in these affections is rather that of an optic neuritis.

IV. The Way in Which Choked Disc is Produced

A. v. Graefe believed that the increase of intracranial pressure caused by the tumor acted to compress the sinus cavernosus, and that this gave rise to an engorgement in the ophthalmic vein, which empties into the cavernous sinus, which was transmitted peripherally through this vein into the central vein of the retina. He thought that this engorgement in the retinal veins induced a serous infiltration and œdema of the papilla, and that the serous transudation acted in turn on the vessels, so that the optic nerve was as it were strangulated in the unyielding network of the lamina cribrosa.

But this theory was rendered for the most part untenable as soon as *Sesemann* showed that the ophthalmic vein has sufficient connection with the facial so that an obstruction to its flow into the cavernous sinus could not produce a choked disc. There are still some investigators, like *Deyl* and *Knape*, who think that the central vein is compressed at a point anterior to its anastomosis with the ophthalmic vein. Such a place exists, according to *Knape*, at the point where the vein passes through the dural sheath of the optic nerve, and, according to *Deyl*, along the passage of the central vessels through the intervaginal space itself.

The theory which has long been accepted as explanatory of the origin of choked disc is the one propounded by *Schmidt-Rimpler* and by *Manz*. Preparatory to this was the discovery by *Schwalbe* that the intervaginal space of the optic nerve communicates with the subdural, or subarachnoid space of the brain. *Schmidt-Rimpler* advanced the idea in 1869 that when there was an increase of the intracranial pressure a large quantity of cerebrospinal fluid was pressed into the lymph space of the sheath of the optic nerve and caused an œdema in the lamina cribrosa, which contains a system of canals connected with the arachnoidal space. The statement of *Schmidt-Rimpler* that a system of canals in the lamina cribrosa communicated with the arachnoidal space was not confirmed by *Leber*, but *Manz* claimed that by means of the hydrops of the sheath the intracranial pressure caused circulatory disturbances in the end of the optic nerve which resulted in an œdematous swelling and infiltration. Since that time this has been known as the *Schmidt-Manz* theory.

Recently *Levinsohn* has tried to prove experimentally that an obstruction to the flow of lymph in the intervaginal space compresses those lymph passages which run in the optic nerve, and that an engorgement of the fluid coming from the vitreous chamber into the distal section of the optic nerve is produced in this way. *Hoche* has proven that analogous changes to those found in choked disc are also to be met with in the posterior roots of the spinal cord. He considers it very probable that in consequence of an engorgement of lymph the optic nerve, which is saturated with œdema, suffers a constriction in or close to the lamina cribrosa.

Another theory based on a purely mechanical origin of choked disc was advanced by *Parinaud*, who claimed that it is present in the case of a tumor only when the latter is complicated by hydrocephalus and œdema of the brain. As the optic nerve can be looked upon as a projected portion of the brain he believed that an engorgement of lymph takes place within it also. According to *Ulrich* likewise choked disc is an œdema due to engorgement localized in the optic nerve, and *Kampferstein* also explains choked disc as an œdema coming from the brain which is pressed into the optic nerve and through the lamina cribrosa into the papilla.

All of these theories ascribe choked disc to a mechanical origin, and whichever one is accepted there is no doubt that it explains certain important clinical features, such as the high degree of the swelling, the change in the position of the lamina cribrosa, which is arched forward, and the enormous œdema. A mechanical origin seems particularly plausible at first glance when we see a choked disc decrease considerably after a trepanation of the skull and after a reduction of the intracranial pressure, and yet there are factors which cannot be made to harmonize with such a theory.

There are many cases in which hydrops of the intervaginal sheath is absent. More than this, numerous authors have proved that choked disc does not consist pathologically of an engorgement alone, but that distinct signs of inflammation are also present in the papilla, which may frequently be looked upon as primary. Hence it was not long before advocates appeared who questioned the *Schmidt-Manz* theory. *Hughlings Jackson* said that a tumor of the brain acted to a certain degree as a foreign body and excited optic neuritis by its irritating influence. *Gowers* then maintained that the choked disc was produced through products of inflammation. *Leber* was a strong advocate of this inflammation theory. The facts that on pathological examination distinct signs of inflammation were often found instead of a simple œdema and passive hyperæmia, and that an ampullalike protrusion of the distal blind end of the intervaginal space was frequently absent, contributed not a little to cause *Leber* to seek the cause of choked disc in the irritation produced by inflammation. He held that the metabolic products of tumors, which mingle with the inflammatory transudations, acted as irritants and caused the papillitis when they reached the bulbar end of the intervaginal space along with the cerebrospinal fluid. This theory has a large circle of adherents. *Deutschmann* injected agar into the subdural space in living animals, and so became convinced that a simple increase of pressure in the cranial cavity alone caused no disease of the intraocular end of the optic nerve, that choked disc must be an inflammatory affection rather than the result of engorgement, and that the substances which excite inflammation press into the intervaginal space. *Zellweger* likewise thought the cause to be the specific inflammatory property of the cerebrospinal fluid in cases of tumor of the brain. *Elschnig* decided from the results of his pathological studies that choked disc was inflammatory. He considered the neuritis to be primary, and the œdema to be a frequent, but not a necessary, local or accompanying phenomenon. *Liebrecht* emphasized the inflammatory proliferations of the sheaths of the optic nerve and thought the disease started from the pia. *Krueckmann* has defined more accurately the idea of the inflammation in choked disc. He recognizes the favoring action of the cerebral pressure, but thinks that the papillitis is produced by somatogenous and autogenous substances set free by the destruction of the tumor cells. Even a stasis of the metabolic products of the normal tissues in the intervaginal space can excite inflammation.

The strife between the adherents of these two theories has oscillated back and forth up to the present time, but most of us think that neither theory can explain all of the clinical facts. According to *Birch-Hirschfeld* the cases with great œdema and very little infiltration counterindicate the inflammation theory, while the absence of dilatation of the vaginal space, especially in fresh cases, is opposed to the purely mechanical theory. There are therefore a number of ophthalmologists who take a middle ground and ascribe an effect to both factors, the engorgement with lymph and the inflammation, but recently signs have appeared that the balance begins to incline toward a purely mechanical origin of choked disc. The good results obtained by palliative trephining of the skull is not the least important cause of this. It would be strange if some of the symptoms of a tumor of the brain, such as the headache and the vomiting, were to be ascribed to the intracranial pressure and a third symptom, the choked disc, to a different factor, the tumor toxine. Two things in particular have been adduced as direct proofs against the inflammation theory: 1. The fact, which has been established clinically and pathologically, that there are a number of intracranial affections which reduce the space within the skull and give rise to a choked disc, in which no pathological signs of inflammation are present in the head of the optic nerve, is opposed to the *general* acceptance of the inflammation theory. Among these conditions are the choked discs caused by extradural tumors, aneurysms and contusions of the skull. 2. It has been insisted upon recently, especially by *v. Hippel,* that the rapid subsidence of a choked disc after operative intervention cannot be made to harmonize with the theory that it is due to products in the cerebrospinal fluid that excite inflammation. Cases have been observed in which the choked disc disappeared in a remarkably short time after trephining of the skull, or opening of the dura. In such cases the products that excite inflammation must still be able to come in contact with the optic nerve, and *E. v. Hippel* considers it "inconceivable that a severe inflammation caused by toxines should recover rapidly when the same toxines—diminished only in quantity—still continue to act on the optic nerve."

We come now to the reasons which either do not oppose or favor the theory that the increase of the intracranial pressure may be the single factor in the production of a choked disc. They are three in number. First, it has been shown that we can draw no conclusions from the presence or absence of hydrops of the intervaginal space as to the presence or absence of an increased intracranial pressure. Second, no unobjectionable evidence has yet been adduced, according to *v. Hippel,* that a choked disc occurs in connection with an intracranial neoplasm without an increase of pressure. Third, there is no parallelism between the size of the tumor and the increase of pressure. The intracranial pressure may be greatly increased by a small tumor. In spite of all this the theory is not yet accepted that the increased intracranial pressure is the sole cause of choked disc, in accordance with the view of *Schmidt-Rimpler.* The main reason for this is that no pathological condition, which may be considered the imprint caused by the pressure, has yet been found to be constant. Even *v. Hippel* admits that there is no doubt that the *Schmidt-Manz* theory is not applicable to all cases. An experiment performed by *Schieck* to explain the pathogenesis of choked disc is worthy of notice. He investigated mainly the question why the increased pressure acted in the sheath and at the distal end of the optic nerve, and found that in the initial stages the central vein is distinctly compressed within the nerve, while outside of the nerve it is free from signs of engorgement. This does away with the old hypothesis of *v. Graefe* of an obstruction of the venous flow into the cavernous sinus, for, if this hypothesis were correct, an engorgement would be visible throughout the entire course of the vein. The vein is likewise not compressed at the place where it passes through the dural sheath, as was supposed by *Knape,* nor in the intervaginal space, as was thought by *Deyl. Schieck* found further in the initial stage an engorgement of the lymph between the sheaths of the optic nerve. But this little increase of fluid alone cannot suffice to cause a compression of the central vessels and of the bundles of nerve fibres, such as is assumed to exist in the *Schmidt-Manz* theory. *Schieck* discards the inflammation theory in that he is of the opinion that in the initial stage the process does not present the characteristics of inflammation, and that the latter originates as a secondary reaction of the tissues injured by the engorgement. He found regularly in the optic nerve in commencing choked disc the formation of ectatic hollow spaces in the axial groups of nerve fibres near and between the central vessels. These hollow spaces are the distended perivascular lymph sheaths of the vessels. He then refers to the experiment performed by *Levinsohn* in which the optic nerves of animals were ligated within the orbit, sometimes leaving the central vessels free, sometimes tying them off. *Schieck* agrees with *Levinsohn* that two currents of fluid are met with at the level of the entrance of the central vessels through the optic nerve sheath, one of which is centripetal from the eye, the other centrifugal from the brain, both in the perivascular sheaths of the central vessels. *Schieck* enunciates the following theory, based on his findings and on the pathological condition of a marked engorgement in the system of the perivascular lymph spaces of the axial bundles, which I give in his own words. "If it is proven that at the level of the exit of the vessels from the trunk of the optic nerve two currents of fluid are met with in the intervaginal space, one flowing axially in the perivascular lymph spaces of the central vessels from the hilus of the papilla, the other coming from the brain, the main condition for a regulated, common flow of these two currents through the vascular sheaths is that they are under a perfectly even pressure. As soon as the fluid in the intervaginal space, which communicates freely with the cerebrospinal fluid, is subjected to a high pressure in consequence of an intracranial increase, produced for example by a tumor, or a hydrocephalus, the outflow from the cavity of the vitreous must be obstructed. *The result is an engorgement of the lymph coming from the vitreous in the axial cord as the first factor in the production of choked disc,* while the cerebrospinal fluid seeking an outlet in the preexisting path is pressed back and the distention of the lymph space is increased. The obstruction to the outflow of lymph in the axial cord from the hilus of the papilla is soon made visible ophthalmoscopically by a swelling of the tissue, and the picture of venous stasis is added by the compression of the central vessels running in the axial cord. Starting from the axial cord the œdema spreads among the bundles of nerve fibres, produces a destruction and strangulation of them and now a reactive inflammation sets in partly from the products of decomposition of the stagnant lymph, partly from the destruction of tissue. The nerve fibres atrophy while the supporting substance proliferates, and the final result is the so called atrophy after choked disc."

The only point in this hypothesis that has been proved as yet is that perivascular dilatations have been found in the vicinity of the vessels of the optic nerve in the axial cord in a number of cases, but it has still to be demonstrated that this condition is constant. In the second place it must be proved with more certainty that these two currents of lymph actually exist in the sense in which they are present in the hypothesis. Thirdly, it is necessary that it should be demonstrated that these two currents of lymph normally are met with under an actually even pressure. Fourthly, it must be determined whether any support can be found in the intraocular change of fluid in cases of choked disc for the assumption that the path of the lymph coming from the vitreous is occluded. Fifthly, it must be learned in how far the pathological changes harmonize with this hypothesis when the overpressure in the intervaginal space is not the governing factor, and the outflow of lymph along the axial cord is obstructed by increased pressure within the eye. Sixthly, if this theory of a purely mechanical origin of choked disc is correct it must be possible to produce choked disc in monkeys without the aid of a tumor. Seventhly, it must be demonstrated that the cerebrospinal fluid contains no substances that can excite inflammation of the head of the optic nerve. This likewise can be determined by experiment. We hope that the solutions of these problems will be favorable to *Schieck's* hypothesis, for then, at least in the theoretic conception of the pathogenesis of choked disc the desired unity will be attained, and then it can be called the best attempt to explain the way in which the condition is brought about. But I doubt if it will furnish any new, practical method of treatment. As long as the advocates of the engorgement theory themselves admit that it is not explanatory of all cases, and as long as a new theory applicable to all is not positive, I believe that *Leber's* theory of the inflammatory origin of choked disc is justified. But ophthalmologists should not speak of toxines in this connection as though the products of metabolism were insufficient to excite inflammation without assuming such a character. I can only admit with certain limitations the conclusion that the rapid involution of choked disc contraindicates the presence of products that excite inflammation, for such a rapid involution is extremely problematical in not a few cases, and we do not know in how far the paths for the outflow of the cerebrospinal fluid which were occluded are rendered free again by the reduction of the intracranial pressure. As it is certain that in many cases of tumor death occurs not simply in consequence of the masses of cells, but as a result of the grave alteration of the general metabolism, it seems to me hazardous to deny the ability of intracerebral tumors to form abnormal products of metabolism. It is beyond our power to judge in how far the cerebrospinal fluid in cases of tumor of the brain differs chemically and biologically from the normal. The substances that cause *Wassermann's* reaction can be demonstrated in the cerebrospinal fluid of syphilitics, though they are absent in healthy brains, and it seems to me that there is scarcely a doubt that the composition of the cerebrospinal fluid is abnormal in cases of commencing tumor of the brain. Aside from the serological peculiarities that I have met with in it, I have established distinct inflammatory symptoms in both papillæ of a monkey in which I allowed fresh, sterile cerebrospinal fluid from a case of tumor to act for a long time under high pressure, while physiological salt solution produced no effect on a control animal. Unfortunately the parallel experiment with normal human cerebrospinal fluid could not be made. As no conclusion can be drawn from one such experiment the question as to the part taken by substances in the cerebrospinal fluid of patients suffering from tumors of the brain which act to excite inflammation specifically upon the optic nerve remains to be demonstrated. But, although further points for the support of the inflammation theory may be discovered in the future, this plays only a secondary part in the explanation of the clinical symptoms in most cases of choked disc. While, therefore, we must not lose sight of it completely at the present time, I am inclined to agree with many other ophthalmologists on a middle ground: Lymph engorgement in consequence of an intracranial increase of pressure, according to the *Schmidt-Manz* theory, or its substitute hypothesis, can take place together with the action of products that induce hyperæmia and inflammation, and cause the different degrees of optic neuritis and choked disc.

V. Treatment of Choked Disc

It is always extremely difficult to localize a tumor of the brain, and this can be done with absolute certainty only in rare cases. When the situation can be made out with a high degree of probability two things have to be taken into account. First, that the cases are increasing in number in which the presence of a tumor in the brain has been indicated not only by a choked disc, but by other symptoms as well, and yet have recovered spontaneously. *Nonne* and *Reichardt* in particular have reported such cases. Second, the question must be raised in every case whether the tumor is not of a syphilitic nature. *Wassermann's* test must be made in every case, and a very energetic antisyphilitic treatment may be instituted at once. If the tumor does not react to the specific treatment, or if syphilis can be positively excluded, and if it is not possible to accurately localize it, we must try to preserve the patient's vision till the end of his life. This may be done by reducing the intracranial pressure, as surgical experience has shown. *E. v. Hippel* has collated from the literature on the subject evidence which shows that the outlook for the preservation of the vision is most favorable when the operation is performed early. The only question is what form of intervention to advise when a radical operation cannot be performed. A practical point is that lumbar puncture is not only ineffective, but may be dangerous in cases in which the presence of a tumor is probable. Improvement of the choked disc may be obtained by lumbar puncture only when it is due to obstinate syphilis, serous meningitis, or injury of the skull. The skull should be trephined in cases of tumor of the brain, for it has been proved that this operation improves the chances for the preservation of the vision. Life may be prolonged and amaurosis warded off for a long time by its means. Experience shows that perfect recovery indeed may follow the operation in cases of chronic hydrocephalus, pseudotumor, and serous meningitis when no tumor is really present, while if it is not performed the patient may be rendered blind even though he recovers from the disease. The general condition usually improves immediately after the trephining, the headaches and vomiting cease. According to the statistics given by *Finckh* the choked disc underwent involution in 30 out of 31 cases. The results given by *Saenger* are similar.

The place to be selected for the application of the trephine must depend on the individual case. *Saenger* recommends the region of the right parietal lobe because prolapse is least to be feared at this point. Palliative trephining has its dangers, and the conscientious physician must take these as well as its advantages into account. How it is to be performed, and whether on one side or both, we leave to the surgeon. Aside from the dangers there are some other objections to this operation. Cases are met with in which choked disc undergoes involution of itself; this is particularly true when syphilitic neoplasms are present. On the other hand an obstinate gumma may be an indication for a palliative trephining when the danger of blindness is very threatening. The objection raised by neurologists, and to be expected in any given case, that after this operation it is impossible to localize the seat of the tumor, because the symptoms are obscured by the reduction of the intracranial pressure, I cannot admit. The main point is that the dangers attendant on this operation are decreasing. It is the duty of the surgeon to decide when some other operation should be performed instead. *v. Bramann*, in conjunction with *Anton*, tried the so called puncture of the corpus callosum, by which an open connection is made between the ventricle and the subdural space, so as to provide an outlet for the cerebrospinal fluid. *Payr* has drained the ventricle in hydrocephalus. Our therapeutic duty is clear. When the tumor cannot be localized and a radical operation is impossible we can at least try to save the vision of the patient by a palliative operation, for, aside for the amelioration of the general symptoms, these patients bear their troubles better and more easily when they can not only feel but see the hand that cares for them.

The fundus should be examined in every case in which the patient complains of a long persistent headache. This is neglected too often. It is not necessary that a physician should be a specialist in order to recognize the signs of engorgement or of inflammation of the optic nerve. When such a condition is met with it will at least lead one to think of cerebral disease, which perhaps has hitherto made itself manifest only by uncertain symptoms, and when a radical extirpation of a tumor of the brain is impossible the beneficial effect that may be produced by a palliative trephining, or by some similar operation, should be borne in mind.

XVIII

Diseases of the Retina

177. The Normal Anatomy of the Retina and the Way in Which to Make a Diagnosis of a Retinal Disease

It happens very often that we are able to detect with the ophthalmoscope a disease of the retina through which the patient first becomes aware of a pre-existing general disease. Aside from simple detachment and glioma almost all affections of the retina are local phenomena of general diseases, and hence the pathological changes to be seen in the fundus are important chiefly because of their diagnostic value.

I. Normal Anatomy of the Retina

The retina lies loosely on the chorioid, adherent neither to it nor the vitreous, and firmly attached at only two places: at the papilla, where the optic nerve fibres pass into it, and at the ora serrata, where it is joined to the subjacent chorioid. In the healthy eye it is held in position upon the subjacent tissue by the pressure of the vitreous, to which is to be added the power of cohesion between its outer layer and the pigment epithelium, to which *Halben* has called attention.

The pigment epithelium belongs embryologically to the retina. It consists of a single layer of hexagonal cells and is firmly attached to the subjacent chorioid in the normal, developed eye. This explains how it is that the pigment epithelium remains attached to the chorioid when the retina is detached. Still this epithelium is also attached to the retina. Its cells send fine offshoots that resemble cilia between the rods and cones, so that the latter dip, as it were, into the cells. These offshoots contain the fine, brown retinal pigment.

The human retina is about $\frac{1}{3}$ mm thick. If we spread it out on a glass, an area to the side of the place of entrance of the optic nerve is to be distinguished by a slightly yellowish color; this is the macula lutea. We cannot see this color in the living eye under ordinary conditions, the subjacent tissue gives it a rather dark hue. We find a little dimple in the centre of the macula, the fovea centralis. The acuteness of vision depends on the functional activity of this area.

The microscopical anatomy of the retina is extremely delicate. We find the following layers from without inward: 1, The layer of rods and cones; 2, the membrana limitans externa; 3, the outer granular layer; 4, the outer plexiform layer; 5, the inner granular layer; 6, the inner plexiform layer; 7, the layer of ganglion cells; 8, the layer of nerve fibres; 9, the membrana limitans interna. The first three layers together form the neuroepithelial layer, or the sensory epithelium, the next six the cerebral layer. To these is to be added 10, the pigment epithelium. It is to be seen at once that there are three different layers of cells in the retina, the outer granular, the inner granular, and the ganglion cell layers. Between these lie numerous fibres which pass from within outward and act as a supporting substance. These are *Mueller's* supporting or radiating fibres and are nothing else than elongated glia cells. The nucleus of each of these cells lies in the inner granular layer, while the cells proliferate outward to the limitans externa and inward to the inner surface of the retina where they form the membrana limitans interna.

We know very imperfectly the physiological parts taken by all these layers of the retina. Our only certain knowledge is that the light perceiving elements are the rods and cones, which acquire the necessary substances from the subjacent pigment epithelium and the choriocapillaris. This layer of pigment epithelium has been compared not inappositely to a photographic plate which is always renewing itself. Of the photochemical processes that take place we know only the following facts: When a retina is kept in the dark a coloring matter accumulates in it that is extremely sensitive to light, and bleaches rapidly under its influence the visual purple discovered by *Boll*. The pigment in the pigment epithelium is continually changing its place; in the dark it tends to accumulate about the nucleus of the cell, but as soon as light enters the eye the granules of pigment press into the processes of the pigment epithelium that lie between the rods and cones so as to envelope these light perceiving elements. The cones of the retina contract in the light and lengthen in the dark. These facts are by no means sufficient to give us an insight into the delicate processes that take place, but one thing is evident: the first process of vision must take place in the outer layers of the retina. The light must pass through the entire thickness of the retina before it can reach the elements by which it can be perceived. The stimulus is guided back again from this point through the layers of the retina and is transmitted by its innermost layers through the optic nerve to the brain.

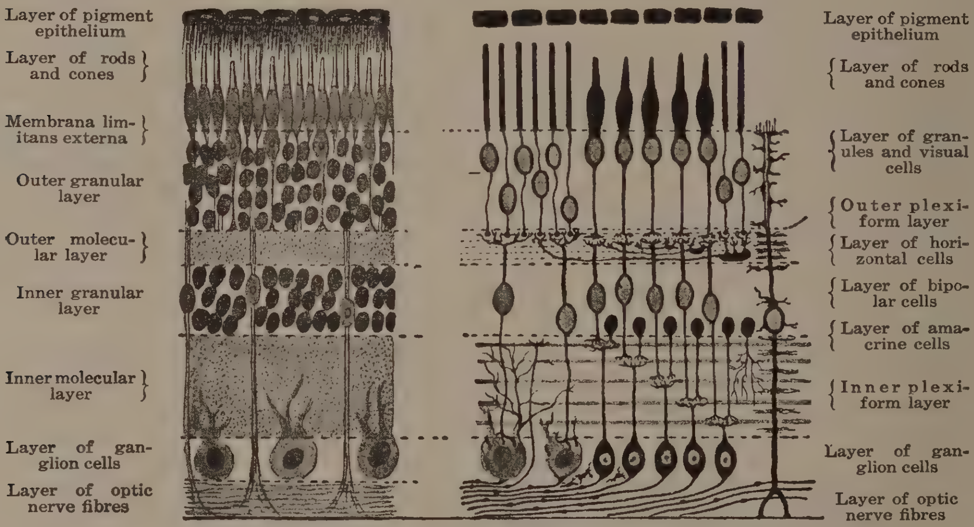


FIG. 134.—Anatomy of the Retina, after *Greef.*

The rod cell is divided into 1, the outer segment; 2, the inner segment; 3, the rod fibre which passes through the external limiting membrane; 4, the nucleus, contained in the outer granular layer; 5, the terminal head, which ends in the outer part of the outer plexiform layer. The plexiform layers, which were formerly known as the outer and inner molecular layers, consist of fine, very dense plexuses of nerve fibres.

In the same way a cone cell is divided into 1, the outer segment; 2, the inner segment; 3, the cone fibre; 4, the nucleus, which is to be found just in front of the external limiting membrane; 5, the foot of the cone, from which fine terminal fibres extend and are lost in the outer plexiform layer.

The second layer, the internal granular, consists of bipolar nerve cells, the outer processes of which connect in the outer plexiform layer with the ends of the visual cells, while their inner processes connect in the inner plexiform layer with the processes of the layer of ganglion cells.

The third layer, that of the ganglion cells, sends its axis cylinder processes as optic nerve fibres in the layer bearing that name to and along the course of the optic nerve. This layer of nerve fibres is composed of naked axis cylinders which do not, as a rule, receive medullary sheaths until they have passed through the lamina cribrosa. Medullated nerve fibres are seldom met with in the human retina.

If we start now from the optic nerve and trace the nerve fibres as they spread out in the retina we find that those which run upward and downward divide and then curve about to surround the macula, while those which supply the medial side of the retina take a radiating course.

In the region of the equator the retina suddenly diminishes to a single layer of high cylindric cells; this is the ora serrata. The part of the retina lying on the ciliary body is formed of only a single layer of cylindric cells, beneath which is the pigment epithelium. Farther forward the cylindric epithelium becomes flatter, and on the posterior surface of the iris the pigment layer and the pars iridica retinæ form only a flat lamina composed of two layers of cells, both of which end at the margin of the pupil.

The macula is a transversely oval area about 4 mm to the outer side of the papilla with a horizontal diameter of 1.8 to 2 mm. Toward the fovea centralis the inner layers of the retina grow thinner so as to form a deep depression. The neuroepithelial layer in the fovea is composed wholly of cones. The rods appear first on the margin of the fovea and constantly grow more numerous toward the ora serrata. The fovea is composed of cones evidently for the perception of minute disparate points.

The central artery of the retina is a branch of the ophthalmic, which in turn comes from the internal carotid, so the retinal belong to the system of cerebral arteries. The central vein of the retina discharges its blood through the ophthalmic vein into the cavernous sinus. The large vessels of the retina lie in the layer of nerve fibres just beneath the internal limiting membrane and cause some of the nerve fibres to bulge toward the vitreous; they then break up into capillary networks which extend in the retina only to the outer limit of the inner granular layer. The entire neuroepithelial layer is destitute of vessels.

II. The Way in Which to Make a Diagnosis of a Disease of the Retina

The retina of the living, healthy eye is perfectly transparent, so all that can be seen of it with the ophthalmoscope is the vessels and the blood contained in them. The only place where the retina reflects light a little more strongly is in the vicinity of the head of the optic nerve, where its thickness is greatest. The inner layers derive their nutrition from the central artery of the retina, the outer ones from the choriocapillaris of the chorioid. This fact makes such an impress on the diseases of the retina that we divide them clinically into two groups. Both vascular regions may be involved, but in such a case one or the other usually preponderates.

The Most Important Diseases of the Outer Layers of the Retina and Their Symptoms

It is self evident that all diseases of the retina, whether of the outer or of the inner layers, cause disturbances of vision, but those of the outer layers cause disturbances that are so characteristic in many cases that the differentiation can be made from them.

In the first place such patients may complain of *flitting subjective sensations of light*, such as rings, discs, sparks and flashes, which may increase with every excitement and are to be ascribed in part to circulatory troubles in the chorioid. This patient tells us that the photopsias become more marked when a bright light suddenly strikes his eyes after he has remained in a dark room for some time. Local disturbances of adaptation play a part in the production of such forms of photopsia. The outer layers of the retina are the ones which are principally irritated by inflammatory processes, as we have seen in exudative chorioiditis. Little central scotomata may give the patients the sensation of flickering and dancing before the eyes, as for example when clear images alternate with indistinct and vacant places while reading.

Another important symptom of disease of the outer layers of the retina is *metamorphopsia.* This symptom indicates two things: that the region of the macula lutea is diseased, and that the neuroepithelial layer of the retina is affected. The retinal images are distorted. Straight lines appear to be bent or crooked, letters seem to be oblique, wanting in parts, or out of their normal position. Frequently the symptom of *micropsia* is associated with that of metamorphopsia; letters look small. The explanation of this phenomenon is that the light perceiving elements of the retina have been pressed apart so that the image of the object falls on a smaller number of percipient elements. The rods and cones can be pressed apart very easily by a subretinal exudate.

When the disease of the macula spreads, the symptom of a *central positive scotoma* takes the place of the metamorphopsia. First there is a disturbance of the process of adaptation which deprives the affected portion of the retina of a part of its sensitiveness to light. The diseased portion feels as if it were less brightly illuminated, and the patients complain of dark spots, i.e., of positive scotomata, in the visual field. It is often necessary to test the field in reduced light in order to detect such scotomata. If the disease of the neuroepithelial layer extends farther into the retina the central area finally loses even the last trace of light perception and the scotoma becomes absolute.

We understand by a *ring scotoma* a more or less closed defect in the field of vision which surrounds the preserved central portion, while outside the ring is another zone in which the vision is intact. We meet with such ring scotomata in the chorioretinitis due to progressive myopia, in syphilitic changes in the fundus and in retinitis pigmentosa.

The most common symptom of disease of the outer layers of the retina is hemeralopia. Vision becomes worse as dusk approaches because the sensitiveness of the retina to slight intensities of light is diminished. The retina is not only insensitive to weak light impressions, but is more dull than a healthy one to bright illumination and adapts itself more slowly to weaker degrees of light. We suppose that in such a condition the visual substances contained in the neuroepithelial layer are replaced more slowly than they should be. Such a disturbance in the metabolism of the retina may be revealed by very different conditions. I have spoken already of the epidemics of hemeralopia during the great fast in Russia. This symptom may appear in general conditions of weakness, in intoxications, as in jaundice, and in the course of such infectious diseases as cause a change in the composition of the blood, like malaria. Hemeralopia is mainly due, however, to diseases of the outer layers of the retina, when it is characterized by a reduction of the color sense in daylight, much greater reduction of the central vision in reduced light than takes place in healthy eyes, and an abnormal contraction of the fields of vision for white and for colors as darkness increases. When these symptoms are present or have been complained of, we must think at once of disease of the outer layers of the retina.

The surest indication of a disease of these layers is furnished by the ophthalmoscopic examination. The phenomenon by which these diseases are distinguished is a pathological development of pigment in the retina. It is supposed that the pigment epithelium derives its nutrition from the choriocapillaris, and that circulatory disturbances in the latter cause nutritive troubles in the former, which may be completely destroyed, or may show signs of proliferation. The pigment spreads by predilection along the vessels. In rare cases the pathological pigmentation of the retina may be hæmatogenous, the pigment coming from absorbed hemorrhages, but in by far the majority of cases in which a decolorization of the pigment epithelium is met with, together with a pathological pigmentation of the retina, the diagnosis can be made of a disease of its outer layers, and the only question to decide is what the disease may be. The principal diseases that come under this category are: 1, the various forms of chorioretinitis, and, 2, retinitis pigmentosa. These need to be differentiated.

When we look into the eye of this patient we find patches of decolorization with pigmented edges scattered very diffusely over his fundus. The pigment of the pigment epithelium has disappeared over large areas, while in other places it is massed in separate heaps, or it may be arranged in the form of a network. The black heaps of pigment have been pressed into the retina and cover the vessels in some places. In other places, where the pigment epithelium has been destroyed and the chorioid is visible, the vessels of the latter are strikingly altered and appear in the form of white or whitish yellow cords. The retinal vessels course over these diseased places in the chorioid. In fresh cases of chorioretinitis we also find opacities in the vitreous because these exudates break through the retina.

The Most Important Diseases of the Inner Layers of the Retina and Their Symptoms

The ophthalmoscopic pictures presented by diseases of the inner layers of the retina vary a great deal. The principal diseases that have to be taken into account are: 1, retinal hemorrhages; 2, occlusion of the central artery; 3, occlusion of the central vein; 4, white spots in the retina and the various forms of the so called retinitis. For didactic and diagnostic reasons it is desirable to decide first whether the affection is of the outer or of the inner layers when we have to deal with a disease of the retina, and it suffices to say that the symptoms and the ophthalmoscopic picture indicate that this patient is suffering from a disease of its outer layers. Taking this as a starting point we can become acquainted with the various diseases.

178. Pigment Degeneration of the Retina

We have made two tests of the visual field of this patient, one with bright, the other with reduced light, and the difference between them is very striking. In clear daylight the extent of the field of vision is approximately normal, while it is greatly and concentrically contracted in reduced light, or in the dusk.

The patient tells us that his vision has been very bad in the twilight ever since early childhood, and that his condition has gradually grown worse until now he can no longer see to go about alone in the evening, although he can still take his walks in the bright sunshine. This sort of visual disturbance is known as hemeralopia.

With the ophthalmoscope we find that the entire periphery of the fundus is covered with a network of pigmented spots which form very delicate figures. They are stellate, jagged and resemble bone corpuscles. The heaps of pigment are united by their offshoots into a complete network within which larger heaps of pigment may be present.

If we examine this pigmentation of the retina rather more carefully we find two marked symptoms. One characteristic of this degeneration of the retina is that it always begins at a distance from the papilla and macula. In most cases and in the initial stage we find the pigment at the outermost periphery of the fundus, whence in the course of decades it slowly and surely encircles the papilla and macula until it finally reaches their vicinity. Or a zone of pigment may develop in the region of the equator of the globe which gives rise to a ring scotoma, but even then the macula and papilla are exempt. Exceptional cases have been met with in which the pigment started from the papilla and surrounded the macula, following the central vein. This peculiar and characteristic change of pigmentation of the fundus suffices for the diagnosis: pigment degeneration of the retina, commonly called retinitis pigmentosa. How can we decide that this is really a disease of the retina and not a chorioiditis? By means of the second peculiarity of this retinal pigmentation. Close relations exist between this pigmentation and the retinal vessels which prove these masses of pigment to be really in the retina. In many places it can be seen that the pigment is proliferated along the vessels and partially covers them, so that they can be seen as black, branching lines. It must be supposed that the fine branched figures are nothing else than the pigmented walls of the partly obliterated, small vessels and capillaries of the retina. Aside from the pigmentation this disease is characterized by an early and marked contraction of the retinal vessels. They are difficult to detect in the periphery and are very small on the papilla. Signs of atrophy accompany the proliferation of the pigment. Large bright spaces appear together with the pigmentation, in which the pigment epithelium has been decolorized, or has been destroyed, especially when the disease has lasted for many years. In these places the chorioid can be seen to be no longer normal; its larger vessels appear to be white or yellowish, because of a process of sclerosis. Lack of pigment may exist in the intervascular spaces, together with an abnormal heap-ing of pigment, so that the bright sclera shines through. Aside from these typical symptoms we sometimes find colloid deposits on the vitreous lamella in the form of small, lustrous white spots, but it is rarely that they occur at the margin of the papilla.

When the disease is far advanced grave changes may be found in the optic nerve, which loses its normal appearance fairly early. Its margins become indistinct, but the principal change is a peculiar yellowish gray color which is very characteristic. This is the so called yellow atrophy of the optic nerve. The atrophy of the papilla is shown primarily by the vessels. Both arteries and veins are so very small that they cannot be distinguished from each other. The number of vessels likewise decreases and finally they can scarcely be traced a short distance into the retina.

Pathology of Retinitis Pigmentosa

Although we are justified clinically by the appearance of the pigmentation in the supposition that this is a disease of the outer layers of the retina, yet the pathological investigation shows that the chorioid also is involved, and it is indeed rather probable that the disease of the latter membrane is primary. Adhesions exist in many places between the chorioid and retina, and there are changes in both the larger and the smaller chorioidal vessels, the walls of which are hyaline, swollen and thickened. Numerous colloid thickenings of the vitreous lamella of the chorioid are present. The pigment epithelium is atrophic in some places, greatly proliferated in others.

The greatest change is found in the structure of the retina, the nervous elements of which are absent, so that the entire membrane has undergone connective tissue degeneration. The layer of rods and cones is almost completely wanting and only slight traces are demonstrable of the outer granular layer. We find abundant proliferation of connective tissue everywhere. The layer of nerve fibres resists the longest, but as soon as the ganglion cells are destroyed the axis cylinders belonging to them must degenerate. We also find a great hyaline thickening of the vessels of the retina, which are completely obliterated in places.

The pigmentation of the retina comes without doubt from the pigment cells which enter it by proliferation and migration, chiefly at places where adhesions exist between the two membranes. As long as the membrana limitans externa is intact the proliferation of pigment is delayed, but as soon as it is destroyed the pigment cells penetrate even into the innermost layers of the retina. The pigment accumulates by predilection in the lymph sheaths of the vessels in the layer of nerve fibres. In other places the heaps of pigment lie in hollow spaces that have been formed in the atrophic tissue. Thus the pigment degeneration of the retina represents a chronic interstitial proliferation of connective tissue in all the layers of the membrane, in which the nervous elements are destroyed by atrophy and the pigment wanders into the retina from the pigment epithelium.

Clinical Course of Retinitis Pigmentosa

The first symptom is hemeralopia. This may exist for a long time before the contraction of the field of vision in reduced light causes any trouble. The peripheral portions of the retina become less sensitive at first, in harmony with the fact that the pigmentation commences in the peripheral parts in the typical cases, but we must not conclude from this that there is any fixed ratio between the quantity of pigment and the degree of atrophy of the retina. A very marked contraction of the field of vision may be present with a slight development of pigment, and cases are met with in which the disease runs its course without any pigmentation; these we call cases of retinitis pigmentosa sine pigmento. The clinical symptoms are exactly the same as in degeneration of the retina with pigmentation, and there are numerous transitional cases between these two groups. At first the disease causes little trouble aside from the hemeralopia. The patients seldom complain of photopsias, they are more apt to suffer from dazzling. The central vision remains good for many years, even for decades, but the visual field gradually grows smaller. A ring scotoma is met with, according to my experience, more often than was formerly believed. In other cases the contraction of the field of vision is concentric from the first. Gradually a time arrives when the patient is still able to read the finest print in good daylight and yet is unable to go about alone in the evening. The situation of such patients can be easily simulated by looking through two small tubes held in front of the eyes. The patients can only grope about.

Both eyes are affected in the great majority of cases, yet a pigment degeneration in only one eye has been met with. Males seem to be more predisposed to the disease than females. The central vision gradually fails and it is exceptional to find good visual acuity with a high degree of concentric contraction of the field of vision. This can be readily understood, for we have seen that an ascending atrophy of the optic nerve is finally developed which leads to amaurosis.

Etiology of Retinitis Pigmentosa

This is another obscure problem; we only know that the predisposition to the disease is congenital. We meet with cases of *congenital amaurosis from atrophy of the retina*, in which we find immediately after birth only slight ophthalmoscopic changes, but during the first year of life a rarefication of the pigment epithelium takes place and an increasing pigmentation of the retina can be observed. Finally the signs of yellow atrophy appear in the papilla and the vessels become very small.

In the majority of cases the clinical signs of pigment degeneration, hemeralopia and contraction of the field of vision, first become evident during or after childhood. Clinical experience has shown that either consanguinity of the parents or heredity is present in a large percentage of the cases. In others, the development of the disease has been ascribed to infectious diseases, such as congenital or acquired syphilis, when we may suppose that they caused the congenital predisposition to manifest itself. In my opinion our material must be revised anew by the aid of biological serum reactions. *Hess* and I have found substances in the normal serum which are able to destroy the elements of the retina by direct contact. If we float the retina of an ox in salt solution the terminal segments of the rods drop off. If some drops of the serum of the same animal are then added to this suspension of the rods the latter are agglutinated in a manner analogous to that of the agglutination of bacteria. Such substances as lysine, which dissolves the rods, are demonstrable in the serum. These substances are separated from the sensitive elements of the retina in the living eye only by the walls of the capillaries and the pigment epithelium. Further than this we know nothing of the way in which the degeneration of the retina is brought about. The congenital nature of the disease is also indicated by the fact that congenital defects of the nervous system, as well as malformations of the eyes and of the limbs, are often associated with the pigment degeneration of the retina. *Wilbrand* and *Saenger* have met with a status nervosus in most of these cases, which is shown by defective intellectual capacity, idiocy, signs of faulty physical development, epilepsy, deafmutism, or impaired hearing power. Other congenital faults of development that may accompany the disease are microphthalmos, keratoconus, ectopia of the lens, persistent hyaloid artery, and I wish to call particular attention to the frequent occurrence of posterior polar cataract which usually remains stationary. Still other congenital faults that may be met with are polydactylia, pes varus and hare lip.

Prognosis and Treatment of Retinitis Pigmentosa

The prognosis is bad. Occasionally the disease stands still for a long time, but the final result is amaurosis, due to the atrophy of the optic nerve. Therapeutically we are impotent. We may secure a temporary enlargement of the field and improvement of vision by injections of strychnine, after which the disease resumes its progress.

The typical clinical picture is easy to recognize. The hemeralopia, the concentric contraction of the field of vision, the condition in the fundus as revealed by the ophthalmoscope, the beginning of the disease in youth and its slowly progressive course, taken together form so peculiar a symptom complex that it cannot be mistaken, and yet a differential diagnosis must be made.

This pigment degeneration of the retina must be differentiated from disseminated chorioiditis, which is also a diffuse disease characterized by discrete circumscribed spots in which a decolorization of the fundus can be perceived together with black spots of pigment. These spots in chorioiditis are roundish, are scattered about irregularly and blend with each other. The heaps of pigment are more lumpy, not as delicate as those in pigment degeneration, but the main point of difference is that the pigmentation in chorioiditis does not show such marked relations to the retinal vessels. The pigment lies behind the vessels and does not follow along their courses. It is true that these foci may also result in a pigmentation of the inner layers of the retina which may exhibit a great deal of similarity to that of pigment degeneration, but even then the disseminated foci can be recognized. The differentiation may frequently be made by the test of the field of vision. We must also remember that we have a retinitis pigmentosa without pigmentation, in which we have the same symptom complex, hemeralopia, progressive contraction of the field of vision and finally atrophy of the optic nerve. Both forms may appear in the same family, so we must look upon them as the same disease, of which the pigmentation is only one symptom. Further, there are cases in which the disease begins as the *retinitis punctata albescens* described by *Fuchs*, in which we find numerous large and small bright points in the periphery and a characteristic pigmentation subsequently develops, as shown by a case that was under my observation for years. It is of less practical interest to us that very rare cases of congenital pigmentation of the retina are met with in which there is no impairment of the vision. On the other hand it is important that we should know that relations exist beyond a doubt between pigment degeneration and glaucoma which are primarily to be ascribed to arteriosclerosis. There can be no doubt that the sclerosis of the vessels in the eye is a very important factor in the etiology of glaucoma.

179. Retinal Hemorrhages as Diagnostic Guides in General Diseases

Every retinal hemorrhage is simply a symptom of some other disease, and our duty in diagnosis is not complete when it has been recognized. The ophthalmoscope is necessary for its recognition, and this instrument is one that should be used not by the specialist alone, but much more commonly than it is by the general practitioner.

I. Ophthalmoscopic Appearance of Retinal Hemorrhages

When we see a retinal hemorrhage it is natural that we should ask first whether it is of *arterial or venous origin*, but this is a matter of little practical importance. We may assume it to be arterial when it is in immediate proximity to an artery. Very often we are unable to determine whether it is arterial or venous, for its color is apt to change and is dependent on the composition of the blood.

The *form of the hemorrhage* is of more interest. Hemorrhages may be punctate, when we suppose them to be capillary, they may be roundish, splashed, or tongue shaped, or quite a lake of blood may be formed. Their number is likewise extremely variable. The situation in which they are found most frequently is near the papilla, but it is a matter of the greatest importance when the macula is involved, as such hemorrhages cause the greatest amount of visual trouble. Hemorrhages in the macula are common in high myopia and in senile changes due to arteriosclerosis.

Next we need to determine *in what layer of the retina* the hemorrhage is situated. As the retinal vessels lie in the layer of nerve fibres a hemorrhage indicates a disease of the inner layers. Elongated, spindle shaped, or ribbon-like hemorrhages are usually situated in the layer of nerve fibres, have an arrangement corresponding to that of the bundles of nerve fibres, and are apt to look feazed at the edges. When a blood vessel appears to be swollen and spindle shaped the hemorrhage is at least partially in the vessel sheath. A hemorrhage on the inner surface of the retina may distend the limitans interna. When it is of a round or irregular form it is usually situated in the middle or deeper layers into which it may penetrate. The blood can also percolate completely through the retina and spread out between it and the vitreous, so as to form a *preretinal hemorrhage*, which is usually situated in the macula and has a round or oval form that is characteristic. It is only in rare cases that these preretinal hemorrhages are found in other places, as for example, on the papilla. After a while the blood sinks and the disc acquires a horizontal upper line of demarcation. Whitish places appear in the mass of blood which consist of white blood cells or of crystals. The causes of preretinal hemorrhages are injuries, nephritis and anæmic conditions. Sometimes the blood breaks through the hyaloid membrane and enters the vitreous.

II. Further Steps in the Diagnosis

Our second ophthalmoscopic duty is to learn whether the retinal hemorrhage is the only demonstrable change in the fundus, or whether other morbid conditions can be found in the papilla or retina. If we find, for example, a choked disc, or some form of retinitis, we need to pay no further attention to the hemorrhage, for our diagnostic thoughts are immediately guided in another direction. Our duty is to diagnose the disease that is present and to proceed from that.

III. Etiology of Retinal Hemorrhages

When hemorrhages are present without other changes in the retina we have to ascertain their cause. The ultimate cause of such a hemorrhage must be either a congenital, or an acquired fault of the blood, or of the walls of the vessels.

a. Congenital Faults of the Walls of the Vessels and of the Blood

Hæmophilia forms the only congenital predisposition to troubles of this nature.

b. Retinal Hemorrhages Due to Acquired Faults

The most varied kinds of retinal hemorrhages are met with after *injuries*, not only direct solutions of continuity by perforating wounds and after contusions, but also after severe compressions of the thorax, such as are met with in railroad accidents. They are to be found very often in *infants just after birth. E. v. Hippel* found them in no less than 10 out of 24 eyes. They may be caused by the change in circulation that takes place at birth, but more certainly by the increase of pressure to which the child's head is subjected. As a rule, they disappear in a short time without having caused any harm, but it is more than probable that in many cases a subsequent amblyopia is to be ascribed to the injury of the sensitive elements by such hemorrhages, even though no gross lesions can be seen.

Local changes in the eye itself may be productive of retinal hemorrhages. In *glaucomatous conditions* the venous engorgement may cause an escape of blood from vessels that are predisposed to rupture. For instance, if an iridectomy is performed on such an eye the sudden reduction of tension may result in a hemorrhage.

Vascular changes form by far the commonest cause of retinal hemorrhages; in these cases we have to search out the source of the change in the vessels. We have to take into account a number of *toxic conditions*, such as chronic lead poisoning, phosphorus poisoning, carbonic acid poisoning, and poisoning with nitrobenzol, hydracetin, roburit, snake venom, or extractum filicis maris. The changes caused in the walls of the blood vessels by *infectious diseases* are of greater importance than these. Retinal hemorrhages, both in the periphery and at the posterior pole, have been observed repeatedly during the febrile attacks of malaria, in typhoid fever, influenza, and miliary tuberculosis, while they are rarely met with in syphilis, except in combination with other changes, such as papillitis or optic neuritis.

Retinal hemorrhages also occur in the *hemorrhagic diatheses*, in purpura, scurvy, and morbus maculosus *Werlhofii.* They are also met with in *anæmic conditions.* After grave losses of blood from ulcers of the stomach, or from the intestines, we often find hemorrhages in the retina which may be either punctate, or extensive, with impairment of the vision. They are very rarely seen in *chlorosis; Pagenstecher* found them in only 3 out of 246 patients, i.e., in 1½% of the cases. They form, on the contrary, an almost constant symptom in *pernicious anæmia*, one which is of great diagnostic importance. In this disease they are usually situated about the papilla. The white spots that are frequently to be seen in them are not characteristic, as they are often found in hemorrhages from other causes, especially in those of leucæmia. As soon as they reach the macula they produce visual troubles, while otherwise they may readily escape observation, and they may be so great as to destroy the eye. A short time ago I was obliged to enucleate the eye of a man suffering from pernicious anæmia because of a very severe intraocular hemorrhage with glaucoma. Such anæmic hemorrhages may also take place after *infection with anchylostomata. Nieden* found them in 8% of the cases of this disease among miners. The botriocephalus latus can also cause anæmia with retinal hemorrhages. They are very common in *leucœmia,* a disease in which they are usually situated in the layer of nerve fibres.

A form of hemorrhage met with in young people is that known as *recurrent hemorrhages into the vitreous.* A darkening appears in one or both eyes of such individuals without apparent cause. We examine and find an extensive hemorrhage into the vitreous which came originally from the retina. These hemorrhages have certain characteristic features. They occur in young persons, usually between the ages of 15 and 25, chiefly in males, and they recur. The cause is still unknown. Anæmic conditions or tuberculosis may be present sometimes without being responsible for the hemorrhages. The resorption of the blood is much more rapid than that in other forms of hemorrhage into the vitreous, so an entirely different process must prevail. Although formerly the source of these recurrent hemorrhages was supposed to be in the vessels of the ciliary body, the more recent observations of *Fuchs, Scheffels* and others have proved that they come from the veins of the retina. The patients frequently state that they are quite subject to nosebleed. An entirely different class of cases of recurrent hemorrhages into the vitreous is that due to gout and anomalies of menstruation. The prognosis in the hemorrhages of young people is fairly good. Repeated attacks may result in the formation of membranes in the vitreous, retinitis proliferans, or glaucoma, but these complications are more apt to occur in older persons.

All of these causes can be excluded in this patient in whom we find a well marked *arteriosclerosis.* This is the most common of all the causes of retinal hemorrhage and should be the first to be thought of when such a condition is seen in the fundus. The hemorrhages due to arteriosclerosis are met with in four forms: 1, Numerous hemorrhages scattered about in the retina with white spots between them; 2, Hemorrhage particularly in the macula; 3, the so called retinitis hemorrhagica, i.e., thrombosis of the central vein, which is very often due to arteriosclerosis; 4, occasionally, hemorrhages altogether in the vitreous.

Such hemorrhages as we see in this patient may undergo quite a number of changes. They may be resorbed and the larger ones may leave behind white patches, but it is seldom that they cause a pigmentation. In many cases we have much less to do with fresh hemorrhages than with their consequences. In this case of *retinitis proliferans* the papilla is almost entirely covered by bluish white masses that project in part into the vitreous and are composed of an extensive proliferation of connective tissue. We can see in such cases that the masses of connective tissue usually send offshoots into the vitreous. The vessels appear at the margins of the mass. The ophthalmoscopic picture varies, but the masses of connective tissue are almost always connected with the papilla. This retinitis proliferans is to be ascribed to hemorrhages that have become organized into connective tissue.

If I have succeeded in impressing the fact that a spontaneous retinal hemorrhage calls for a general examination of the entire organism, especially of the blood and urine, I have attained my purpose. No special skill with the ophthalmoscope is necessary to detect such a hemorrhage, and its timely recognition contributes to place us on the track of the general disease.

180. Arteriosclerosis in the Fundus and Occlusion of the Central Vein

This aged patient complains that he has suffered for a long time from tinnitus aurium, attacks of dizziness, a feeling of constriction at the epigastrium, palpitation of the heart, loss of hearing, insomnia and weakness of memory. A few days ago he noticed a rapid loss of vision in his right eye. This is a case of retinal hemorrhage due to arteriosclerosis. Whether the age and the associated troubles indicate the presence of this disease in such patients, or whether we arrive at the diagnosis by the exclusion of other causes, we must know how arteriosclerosis of the retinal vessels appears ophthalmoscopically.

Various pathological processes take place in the vessels of the retina. We find a thickening of the walls of the arteries, especially of the intima, and degenerative changes, such as fatty degeneration, sclerosis and calcification of the vessel wall, as well as inflammation and proliferation.

Changes That Are Visible with the Ophthalmoscope in the Vessels of the Retina in Arteriosclerosis

The first sign of arteriosclerosis of the retinal vessels is a *broadening and a tortuosity of the arteries.* Such a tortuosity of the retinal arteries as we find in patients between 45 and 50 years of age is due to a loss of elasticity in their walls. Pulsatory movements within them may be seen in marked cases. The *changes in the walls of the vessels* that are visible with the ophthalmoscope are of great diagnostic significance. Under normal conditions the walls of the retinal vessels are perfectly transparent, so that ordinarily we see only the columns of blood within them, and it is only in the upright image that they may be seen as narrow whitish lines along the sides of the blood columns. One of the most important changes to be seen in arteriosclerosis is a *white margin* to the arteries caused by the thickening and opacity of their walls. This must be differentiated from a whitish sheathing of the vessels which is a congenital anomaly, in which the narrow, lustrous striæ that accompany the vessels are to be considered as continuations of the connective tissue of the lamina cribrosa. Another arteriosclerotic change in the walls of the retinal vessels is that the arteries seem to be transformed into white bands that often appear to be bloodless and can be seen in the upright image to contain only very minute columns of blood. In these cases there is also a hyaline degeneration of the thickened vessel wall.

A third peculiarity is a narrowing of the artery in places, or *circumscribed thickenings of the wall,* when certain parts of the blood columns can be seen to be very small. This contraction of the lumen of the vessels is due to *endarteritis;* an opacity of the vessel wall need not be visible. In rare cases little glistening points may be seen, which are due to deposits of lime or of cholesterin in the thickened adventitia of the arteries.

The symptoms in the veins are quite analogous. The veins may be broad and tortuous, may have white margins, or may be transformed into white cords, and we find in them, even more frequently than in the arteries, circumscribed constrictions of the lumen with nodular swellings of the wall. Sometimes we find the arteries, sometimes the veins to be the more affected, but often the changes in both systems of vessels are uniform. Sometimes the angiosclerosis affects only very circumscribed areas of the retinal vessels, sometimes the changes are confined to one eye. These alterations in the vessels may lead to a series of complications.

Ophthalmoscopic Picture of Thrombosis of the Central Vein

The first symptom is that the boundary lines of the optic nerve cannot be distinguished, because of the cloudy swelling of the tissue, and especially because they are covered by the masses of hemorrhage which form the second phenomenon characteristic of thrombosis of the central vein. The hemorrhages are lumpy or striated, are dark red or black, and usually lie over the vessels. Little round ones may also be seen which hang like grapes in bunches. Between the hemorrhages may be seen in some places gray or whitish opacities of the retinal tissue.

The third symptom is the appearance of the vessels. The arteries seem to be very small, while the veins are extremely dilated and tortuous. The columns of blood are broadened and have a black red color. Sometimes such a vein disappears in a great lake of blood, or is covered by the opaque tissue of the retina.

The question may arise whether it is the main trunk of the vein, or only a branch that is thrombosed; in the latter case the hemorrhages are to be found only in the area to which the branch is distributed.

After the condition has lasted some time the small hemorrhages become absorbed, but changes of color that are visible with the ophthalmoscope gradually appear in the larger ones, caused by the transformations the masses of blood are undergoing. It is rarely that a dark patch of pigment takes the place of the hemorrhage, usually white spots appear in the middle of the extravasation of blood, which are formed by the white corpuscles, and delicate formations of crystals may also be observed. Anastomoses form in the course of the thrombosis, and occasionally suffice for the preservation of a little vision. These anastomoses form delicate convolutions of vascular loops, which remind one by their appearance of the glomeruli of the kidney, situated usually on the papilla, but sometimes in the retina itself. Such massive hemorrhages can appear, according to *Harms*, only when the branches of the vein are still relatively healthy in comparison with the occluded trunk, which opposes only a little resistance to the engorging blood. If the walls of the branches of the vein are sclerosed and thickened the veins have previously become thinned and the hemorrhages have more the appearance of splashes.

Such an occlusion of the central vein may be a marantic thrombosis, in which the thrombus is formed in a lumen which previously was freely open, or it may be due to a primary disease of the vessel wall in the form of an endophlebitis, which by its proliferation causes occlusion of the vessel. The disease of the wall of the vein need not be of high degree, an insignificant injury of the endothelium may suffice to cause a thrombosis.

Thrombi in the central vein may be situated in the retina, in or behind the lamina cribrosa, or farther back, at some distance from the eyeball. The formation of thrombi is naturally favored when obstructions to the flow are present in the venous system.

The prognosis of occlusion of the central vein is bad, an improvement of the vision has seldom been observed. More often new hemorrhages appear, when the patient believes from his subjective sensations that he is improving. Glaucomatous conditions supervene and destroy the last trace of vision in quite a few cases. Occlusion of the central vein is met with chiefly in old people who are suffering from arteriosclerosis, heart disease, hypertrophy of the left ventricle, and emphysema of the lungs. We can do nothing more than to confine ourselves to the general treatment of the arteriosclerosis.

181. Occlusion of the Arteria Centralis Retinae

When this patient woke up yesterday morning he discovered that his right eye had become totally blind during the night, although he had never known it to be diseased.

We find the right optic nerve to be abnormally pale. The larger arteries have become slender threads, the smaller ones cannot be seen. The veins on the contrary are rather small only on the papilla itself. The entire retina about the papilla, as far as the macula and beyond it to the peripheral parts of the fundus, appears cloudy and milky white. Another characteristic phenomenon is a bright, almost cherry red spot in the macula. This is the typical picture of an occlusion of the central artery of the retina.

When the central artery is obstructed either an insufficient quantity of blood, or none at all, can any longer enter the vascular system, the arteries must appear to be small and the papilla pale, and it can be readily appreciated that such a sensitive and highly organized membrane as the retina will suffer from a disturbance of its nutrition and become cloudy. How the red spot in the macula comes to exist is a question concerning which there has been much dispute. It may be due in part to little hemorrhages, to a retinochorioiditis, and to the yellow pigment of the macula, but there is no doubt that in most cases the red of the chorioid shines most distinctly through the retina which is quite thin at the macula and is made to appear still brighter by contrast with its surroundings. In addition to this the cerebral layer of the retina, in which the cloudiness is situated, is absent at the macula.

It was supposed for a long time that the cause of this clinical picture was an actual embolism of the central artery, but now we know from numerous clinical observations and anatomical researches that a true embolism, by which is meant an occlusion produced by a plug driven into the artery by the circulation, is extremely rare, and that most of the cases originate in a different way. I shall not speak of the cases in which the optic nerve is lacerated by injuries in front of the place of entrance of the central vessels, or in which the central artery is compressed within the trunk of the optic nerve by tumors. Far more often the causes are *thrombotic processes in the central artery* excited by degenerative changes in the walls.

A thrombosis has been observed to extend into the central artery of the retina and to cause sudden blindness after ligation of the common carotid, but in most cases the occlusion is due to a primary *endarteritis proliferans* of the wall of the artery, in which the lumen is contracted by a progressive proliferation of the intima. Although at first the blood continues to be pressed through the narrow lumen, yet occasionally the walls may touch each other when the blood pressure falls, and the current of blood may be interrupted when the tonicity of the vessel is increased.

How can we distinguish clinically whether the case is one of true embolism, or of endarteritis proliferans? We must always determine what indications point to the one cause and what to the other without regard to the rarity of true embolism.

We first note whether the retinal vessels refill with blood soon after the occlusion, as frequently happens. We suppose that the restoration of the circulation is made possible by a retrogression of the endarteritis proliferans.

A point is made against embolism when we cannot find a source from which the supposed embolus could have come. According to *Kern* no cause for the embolus can be found in 66.3% of the cases, so that a local disease in the artery is probable in the great majority. In many such cases no embolus could be found in the subsequent microscopical examination of the eye itself, which is additional evidence that the occlusion was really produced by an endarteritis proliferans. We may also be able to diagnose an endarteritis rather than an embolus when we are able to see that the walls of the vessel are diseased, either just after the occlusion, or at a later time.

Finally, we are compelled to suppose endarteritic proliferations to be present when temporary obscurations of vision, or even blindness, take place with the ophthalmoscopic picture of embolism. I have seen a patient, who had almost total blindness of both eyes with the picture of embolism, in whom perfectly free circulation was restored and the central artery did not become finally occluded until a year later.

According to the researches of *Harms* the diagnosis of a true embolism is justified only when we can exclude a primary disease of the wall, when a defect of the valves of the heart, or some other plausible condition of disease can be demonstrated as its probable or possible cause, and when signs indicative of a thrombotic occlusion of the artery are not present.

We find ourselves in the same position when not the trunk, but a branch of the central artery is occluded. This so-called embolism of a branch is more common than that of the main trunk of the artery. We can usually explain the clinical symptoms just as well by the assumption of an endarteritis proliferans as by that of an embolism. It would seem as though it must be easier to actually see the embolus when it is in a branch, than when it is in the trunk and may be situated within the nerve itself, but often it cannot be decided with certainty whether a certain little yellow spot is to be considered an embolus, a thrombus, or a thickening of the wall. We may now and then suppose it to be a true embolus when we learn, by testing the field of vision, that the function of the affected area is permanently lost, because the arteries of the retina do not anastomose. A case like this calls for an examination not alone of the eye, but of the entire body. Sudden blindness from occlusion of the central artery is one of the many manifestations of arteriosclerosis that we meet with in the eye.

The clinical course varies according to the cause and the duration of the occlusion of the vessel. Occlusion of the central artery of the retina usually leads to total amaurosis of the eye, but cases are met with in which the sudden blindness passes away and a high degree of amblyopia, or an amaurosis, develops later. It may also happen that the vision is completely restored in spite of the sudden blindness. In such cases sufficient room is made by the distention of the vessel, or the retrogression of the occlusion, for a small column of blood to pass and to restore its function to the retina. To these are to be added the cases in which only a portion of the vision is restored after a blindness which is at first total. To understand these cases I must recall to mind that we meet with vessels in the retina which come not from the central artery, but from the system of ciliary vessels. These are the *cilioretinal vessels.* We recognize them by the fact that they bend about like hooks in the vicinity of the papilla and disappear into the retina. In such a case the nutrition of a portion of the retina is derived from these cilioretinal vessels, and the region between the papilla and the macula retains its functional power. Even when these vessels are absent a portion of the vision may be preserved. A branch of the central artery may have been given off above the place where the trunk has become occluded and may pass into the retina at the margin of the optic nerve.

Occlusion of the central artery is almost always confined to one eye; cases in which both eyes are affected are extremely rare. Many operative attempts have been made to reduce the tension of the eye and so to promote the entrance of blood into the arteries, such as paracentesis of the anterior chamber and iridectomy. *Mauthner* recommended massage of the eye, but generally the prognosis of occlusion of the trunk of the central artery is bad.

Elsewhere in the body tissue that is completely deprived of its supply of blood becomes gangrenous, but this is not the case with the retina. The cloudiness of the latter lasts a few days and then undergoes involution, leaving the membrane again transparent, but incapable of function. Atrophy takes place simultaneously with a hypertrophy of the connective tissue. As the cloudiness of the retina subsides the red spot in the macula becomes more indistinct and the normal red of the fundus reappears. The fact that the retina has become atrophic is shown mainly by the papilla, which continues to be pale and finally becomes white, while its vessels are like threads and often appear to be accompanied by white striæ. When opportunity offers to follow such a case systematically noteworthy observations can be made on the retinal vessels. Their fullness may improve some days after the occlusion, or the blood columns may break up into little separate cylinders which make slight rhythmic movements in the direction of the blood current.

182. White Spots in the Retina as Signs of Different General Diseases

This patient tells us that he has always felt well, but the ophthalmoscopic examination has revealed hemorrhages and numerous glistening white spots in his retina, which indicate to us that he is suffering from a serious general disease.

1. Is the Lesion in the Retina or the Chorioid?

Both the retina and the chorioid are involved in many morbid processes, when the changes are said to be chorioretinitic, but it is not difficult, as a rule, to determine in which membrane the lesion is situated when we take the following points into account.

Fresh lesions in the chorioid are not brilliantly white, but yellow or reddish yellow. When they are older they look white because the sclera shows through in consequence of the atrophy of the tissue of the chorioid and of the pigment epithelium, but then we find more or less pigmentation, as a rule, at the margin of the spot. If in addition the vessels of the retina pass over it, its situation is indicated as being in the chorioid. Difficulties can arise only in the differential diagnosis of miliary tuberculosis and sepsis, when round, whitish spots without hemorrhages may be present.

2. Differentiation of White Spots from Medullated Nerve Fibres

Thus we come to the conclusion that the white spots are situated in the retina of this patient. The next question is whether they are pathological. This is answered by the great impairment of vision and the accompanying hemorrhages, but if these were not present the spots should not be mistaken for the *medullated nerve fibres* that are occasionally met with under normal conditions.

The fibres of the optic nerve normally lose their medullated sheaths at the lamina cribrosa, but it happens exceptionally that individual bundles retain their medulla for a distance into the retina. Then we see at the margin of the papilla, usually above and below, white tufts, in the radiating or fibrous striation of which we can recognize the arrangement of the nerve fibres. The optic nerve is not diseased in such a case, as is shown by the fact that its color is normal and its margins sharply defined everywhere that the medullated fibres are absent. The entire papilla is covered by the medullated fibres only in rare cases. The medullated nerve fibres themselves may be distinguished from the morbid white spots indicative of general disease by the fact that they are feazed along their margins, corresponding to the courses of the nerve fibres, and so form a flamelike figure. The vessels of the retina are partly visible between these fibres, partly covered by them. They are of such little consequence that they are never discovered except by accident. At the most they can only increase the size of the blind spot. It is evident that the development of medullated nerve fibres is due to a congenital anomaly, for such eyes frequently display other congenital faults, such as the formation of a posterior staphyloma. It is only under one condition that they can be mistaken for patches of degeneration in the retina, when they first appear at some distance from the papilla. Even then their fibrous appearance, their immutability, the normal condition of the rest of the fundus and the absence of general disease leave no doubt concerning the phenomenon. It may be mentioned as rare occurrences that the medullated nerve fibres have been seen to disappear, by *Wagenmann* in a case of tabetic atrophy of the optic nerve, and by *Sachsalber* in a case of tumor of the brain.

3. Pathology of the White Spots in the Retina

The white spots in the retina that occur in general diseases may be due to various pathological changes. They may be varicose thickenings of the nerve fibres, fibrinous exudates, heaps of lymphoid cells, patches of fatty degeneration of the retinal tissue, or foci of embolic inflammation or of calcification. These pathological conditions cannot be distinguished apart with the ophthalmoscope.

4. Etiology of the White Spots in the Retina

Clinical experience has proved that white spots may occur in the retina in a very large number of diseases, a few of which are poisoning with phosphorus or with quinine, diseases of the liver, carcinoma of the stomach, and hydrocephalus internus. Arteriosclerosis may produce, in addition to hemorrhages, numerous white spots of various forms and sizes. White patches of degeneration are met with in syphilis, sepsis, and in the diseases of the blood, like anæmia. Diabetes and chronic nephritis have to be taken particularly into account. Even a tumor of the brain can excite in rare cases a picture like that of albuminuric retinitis. This versatility in the occurrence of such spots in the retina is due to the fact that the highly organized and easily destructible protoplasm of the tissue of the retina, with its very small capillaries, reacts very readily to circulatory disturbances and is often affected sooner than the other tissues by all possible changes in the composition of the blood and juices of the body. Hence it is that the retina is so often an indicator of constitutional disease.

As soon as the physician discovers white spots in the retina he must not content himself with the examination of the eye, but must investigate the entire organism with special reference to the composition of the blood and the presence of albumin and sugar in the urine. The examination of the urine of this patient reveals the presence of a chronic nephritis, and we have before us the picture of the so-called retinitis albuminurica.

Although the fundamental cause of a disease of the retina can be determined positively only by the examination of the entire organism, we must not content ourselves with this, but rather study closely the changes in the fundus. This we should do for several reasons. Each disease is of interest. Physical knowledge is the guiding star of our science. We often gain important data for the prognosis. In many cases the ophthalmoscopic picture is so characteristic as to form an accurate guide to the diagnosis. The time is certainly past in which it was believed that a disease of the kidneys caused a retinitis albuminurica, a diabetes a retinitis diabetica, a leucæmia a retinitis leucæmica. All of these so called forms of retinitis are nothing else than the ophthalmoscopic impression of anatomical injuries of tissue and disturbances of the circulation in the retina which are caused more or less by a disease of its vessels, and yet we are justified in asking what ophthalmoscopic pictures occur in the individual diseases of the organism in order that we may learn to draw conclusions from them. What diseases chiefly are to be taken into account when we see these white spots? What peculiarities of the ophthalmoscopic picture are so marked that we can obtain diagnostic data from them? Beginning with the rarer and ending with the more common and more important diseases we find that white spots in the retina may indicate: 1, retinitis striata; 2, retinitis circinata; 3, retinitis septica; 4, retinitis syphilitica; 5, retinitis leucæmica; 6, retinitis diabetica; 7, retinitis albuminurica. Before we proceed I wish to say that these designations are not very appropriate, for a true retinitis is present in only a single group, but as these terms have been generally adopted I will retain them.

In the so called *retinitis striata*, a rare affection, we find lustrous or bluish white striæ in the retina that branch frequently and extend over a large part of the fundus. They lie behind the vessels and in front of the pigment epithelium. The ends of the broader stripes are apt to be teased out into fibres and sometimes pass over into gray or strongly pigmented patches. This condition is not to be mistaken for retinitis proliferans, in which the bands of connective tissue formed from hemorrhages in the retina and vitreous lie for the most part in front of the blood vessels and extend into the vitreous.

Next comes the degeneration of the retina described by *Goldzieher*, as well as by *Hutchinson*, and named by *Fuchs*, *retinitis circinata*. This is a rare disease which lasts for years. A ribbonlike zone is to be seen in the vicinity of the macula, composed of larger and smaller white plaques, over which the vessels pass. The optic nerve shows no signs of inflammation, but hemorrhages are not uncommon. The disease may affect one or both eyes, it gives rise at an early stage to serious visual disturbances, especially to a central scotoma. The cause is still unknown. Perhaps it may be associated with arteriosclerosis, but as pathological investigation has shown that the degeneration of the retina is caused by hemorrhages and fatty degeneration in a way quite analogous to that in which retinitis albuminurica is produced, and especially as the picture may closely resemble that of the latter, renal disease must be thought of and excluded.

In septic diseases it may happen that the agents of inflammation are carried by way of metastasis into the vessels of the retina and there excite suppuration. Then we have a metastatic ophthalmia. By *retinitis septica* on the other hand we mean a clinical picture in which white patches are found together with hemorrhages in the retina. These white spots are roundish or oval, are usually situated close to the papilla, have little relation to the large vessels of the retina, and are accompanied by hemorrhages. This septic retinitis may occur in two ways: The altered composition of the blood in septic diseases may bring about changes in the walls of the retinal vessels which in turn may result in hemorrhages and degenerative processes in the retina. In this form there is no bacterial contagion, but only a toxic impairment of the nutrition of the retina. In the other form, which presents the same ophthalmoscopic picture, emboli of bacteria are found which excite circumscribed inflammations and do not lead to the development of metastatic ophthalmia. The virulence of the inflammatory agents is slight, so that the foci of inflammation remain localized. I would not dwell upon this septic retinitis if it were not that a differential diagnosis needs to be made at times between typhoid fever, miliary tuberculosis, tuberculous meningitis, and sepsis. In miliary tuberculosis the foci in the chorioid are not white, but rather yellowish, in tuberculous meningitis optic neuritis forms the important symptom, and when we have to differentiate between typhoid fever and sepsis the retinal hemorrhages indicate the latter, for they are extremely rare in the former disease.

It is a fact to be borne in mind in practice that syphilis alters the vessels of the retina in a manner analogous to that in which it changes the vessels of the brain. In *retinitis syphilitica* the vessels are either thickened as a result of perivasculitis, or an inflammation of all the coats of the vessel takes place and results in a proliferation of connective tissue. The capacity of the vessels may be greatly diminished, especially by a proliferation of the intima. They then appear ophthalmoscopically to be transformed into white stripes, or the veins may have white edges. If the vascular changes are situated mainly in the chorioid a syphilitic disease develops at the same time in the retina, chiefly in its outer layers, forming a chorioretinitis syphilitica. Syphilitic disease of the central vessels may cause changes in the inner layers of the retina in which we find hemorrhages, white spots and œdema. If the syphilis has also attacked the vessels of the kidney the syphilitic retinitis may resemble exactly albuminuric retinitis with the stellate figure in the macula, but usually the picture is different. We find a diffuse serous infiltration of the retina in the vicinity of the papilla, where the former appears gray and cloudy, making the margins of the latter indistinct. In the midst of this infiltration lie hemorrhages and discrete yellow foci, which are considered to be infiltrates and accordingly establish themselves by predilection on the vessels. The diseases of the walls of the arteries are particularly to be noticed in syphilitic retinitis. Sometimes the blood column can no longer be seen at the places where the wall is thickened; in other cases little white scales can be seen on the arteries. Frequently this luetic retinitis causes dustlike opacities in the vitreous. Under proper antisyphilitic treatment these exudates may become resorbed from the retina. In graver cases the pigment epithelium becomes involved. The retina may become atrophic and the increasing pigmentation may give it an appearance similar to that of pigment degeneration.

The anæmic condition of the patient may suggest those forms of retinitis that accompany diseases of the blood. Hemorrhages are found in the retina, almost as a rule, in pernicious anæmia, but white spots occur more rarely. These degenerations are sometimes met with in general cachexia. The worm disease of diggers and brickmakers produces an anæmia which results in such diseases of the retina, but *leucæmia* is the principal disease which is associated with such changes. In this disease we very often find white spots with red margins and sometimes the fatty looking spots that are to be seen in albuminuric retinitis; in leucæmia these consist of varicose nerve fibres, of retinal elements that have undergone fatty degeneration, and of infiltrations with lymph cells. Sometimes the fundus appears to be of a bright yellow color; the cause of this has not yet been learned. The great reduction in the amount of hæmoglobin in the blood cannot always be the cause, for the fundus may appear to be of its normal color when the amount of hæmoglobin is very small. *Stock* has found that in many cases lymphomata of the chorioid and accumulations of leucocytes may give rise to a yellow color of the fundus in myeloid leucæmia. As changes in the macula which resemble those of albuminuric retinitis have been observed in leucæmia it is evident that we have to take such changes in the blood into account in the differential diagnosis of white spots in the retina.

A disease which must be thought of in every case in which we meet with white spots associated with hemorrhages in the retina is diabetes. The picture of *retinitis diabetica* is characterized by little lustrous white spots that are accustomed to lie in the vicinity of the macula, between which hemorrhages are to be seen, but the pictures presented may be manifold. White spots may be present without hemorrhages, or hemorrhages without white spots. Even a stellate figure in the macula is sometimes to be seen, but this is rare in diabetes and therefore has a certain diagnostic value. The papillomacular bundle of fibres is often particularly affected and then a central negative scotoma is present. A point of value in the diagnosis is that retinitis diabetica is rare and occurs chiefly in the worst forms of diabetes, when, as a rule, the fundamental disease has already been recognized. The hemorrhages generally preponderate in this form of retinitis.

When we consider the ophthalmoscopic picture of *retinitis albuminurica* we find four cardinal symptoms. First, the retina appears to be cloudy, especially in the vicinity of the optic nerve, where it is thickest, so that the margins of the papilla are rendered indistinct and the vessels are obscured. This cloudiness may be uniform, or it may be finely striated. Changes may also be perceived in the walls of the vessels.

The second cardinal symptom is formed by the hemorrhages which lie in the vicinity of the papilla and are either punctate or linear.

The third and most important symptom is formed by the white spots which are lustrous and have various forms. In many cases they are confluent and then the papilla may be surrounded by a large, lustrous, white surface. When the disease has lasted a long time the white spots may be spread diffusely over the entire fundus.

The fourth characteristic symptom is the stellate arrangement of the white spots in the region of the macula. The rays of this stellate figure are composed of larger and smaller little white marks and converge toward the macula. The centre of the stellate figure looks red brown by contrast. Sometimes the rays are broader and more irregular and may form quite a large white surface.

The patients complain of a cloud before their eyes, and this disturbance is always present in both eyes, though it may differ in degree. One consolation is that the patients usually retain sufficient vision to enable them to go about alone. Total blindness is scarcely ever caused by albuminuric retinitis itself. Careful investigation of the vision of such patients frequently reveals hemeralopia, or a disturbance of the color sense, such as violet blindness, or blue blindness. Another characteristic feature is that improvements and deteriorations of the vision continually alternate. The degree of visual disturbance is not proportional to the changes in the fundus. It is not at all infrequent for a detachment of the retina to be added to an albuminuric retinitis, and an apoplexy of the brain causing homonymous hemianopsia may take place in the course of the disease.

The entire clinical picture depends on a hyaline degeneration of the vessels, met with chiefly in the small and smallest arteries and capillaries. The entire wall of the vessel is transformed into a hyaline mass by which constrictions and even total obliterations of the lumen are produced. It is easily conceived that nutritive disturbances in the tissue of the retina can be brought about in this manner. Numerous hemorrhages are present in the anterior layers of the retina, and in the intermediate granular layer numerous spaces may be seen between the fibres which indicate an œdema of the retina. Most of the white spots consist partly of fibrinous masses of exudate, partly of varicose nerve fibres, which frequently are pressed closely together, and of wandering cells that have undergone fatty degeneration, fatty granular cells. These fatty granular cells appear mainly in the two granular layers, but may be found in the inner reticular layer and in the layer of nerve fibres. The supporting tissue may likewise appear as if studded with minute drops of fat. The fluid that infiltrates the retina may also coagulate into colloid masses in the hollow spaces. The radial fibres likewise exhibit a homogeneous appearance. The layer of rods and cones is destroyed. All this is presented to us in the ophthalmoscopic picture as a white spot. In addition, grave changes take place in the vessels of the chorioid.

This characteristic ophthalmoscopic picture is met with chiefly with the contracted kidney, in *Bright's* disease, but every contracted kidney does not produce this condition; the figures given vary, but it is present in between 10 and 25% of the cases. The connection that exists between diseases of the eyes and of the kidneys forms an important biological problem. Probably the injury to the vessels and the tissue is to be ascribed to cytotoxic substances. Albuminuric retinitis is also met with in many other forms of renal disease, as in amyloid degeneration of the kidney, in the nephritis due to chronic lead poisoning, in that of pregnancy, and in that which follows scarlet fever.

Clinical experience has shown that the prognosis in the two forms last mentioned is much better than it is in chronic contracted kidney. After scarlet fever and pregnancy this retinitis may undergo involution, provided that a chronic nephritis does not develop. We must suppose that the vascular changes which take place in pregnancy and scarlatinal nephritis are of a temporary nature. The prognosis of retinitis albuminurica associated with contracted kidney is quite different. Numerous statistics have shown that death is to be expected within one or two years after its appearance, often within a shorter time. It is only in exceptional cases and under favorable conditions that the patients live longer than this, and therefore it becomes the duty of the physician to acquaint the relatives of the patient with this fact as soon as the diagnosis of an albuminuric retinitis has been established. The sight of an albuminuric retinitis with the ophthalmoscope is a sentence of death in such cases; the patient stands at the gates of eternity and we can do nothing more than to make his last days as easy as possible by proper treatment. Nothing can be done for the eyes.

183. Senile Change of the Macula Due to Arteriosclerosis of the Central Part of the Retina

When quite elderly people complain of an impairment of vision for which no plausible explanation can be found it is well to remember that a senile degeneration may take place in the macula and be responsible for the loss of vision. We cannot content ourselves with an ophthalmoscopic examination of the papilla in such cases, for there is a wonderful contrast between the high degree of visual disturbance and the insignificance of the changes to be seen in the fundus.

We find in the macula of this patient a rose colored or yellowish red spot with a slight increase of pigment. In other cases little whitish places with minute crystals are to be seen, or there may be only a bright or dark speckling. When a high degree of degeneration is present the yellowish spots may form larger, lobed figures. The rest of the fundus may be perfectly normal, or the disc of the optic nerve may show a slight senile paleness with sclerosis of the vessels.

The pathological examination of such eyes reveals an atrophy of the layer of cones, a softening and atrophy of the pigment epithelium and partial obliteration of the choriocapillaris, processes which are to be referred ultimately to arteriosclerosis. Insignificant disturbances of nutrition are evidently sufficient to affect the delicate cones.

In making the diagnosis we have to differentiate the colloid formations which occur on the vitreous lamella in the region of the macula, and must not be mistaken for this disease. The colloid formations are to be recognized from their position behind the vessels, their round form and their brilliant color. Therapeutically we are impotent in senile degeneration of the macula. It is a disease which sometimes takes us by surprise in an unpleasant manner in patients from whom cataracts have been extracted by operations that have been faultless in their performance, but whose subsequent vision leaves everything to be desired.

184. Hereditary Syphilis in the Fundus

The changes in the fundus met with in hereditary syphilis present various forms. In this patient we find a pigmentation of the retina in the peripheral portions of the fundus, but the pigment is not arranged in figures resembling bone corpuscles, the fundus looks rather as if it had been sprinkled with snuff. Together with this delicate pigmentation we find a peculiar yellow red speckling. But the picture may be quite different. We find in the periphery bright, elongated or round, yellowish spots, with heaps of pigment in their centres, particularly in cases in which the disease is associated with interstitial keratitis. The black or gray pigment spots in the brighter places may be entirely wanting and then we find only whitish places which are round in form and frequently coalesce. The pigment epithelium and the chorioid are quite destroyed in these places. We may thus be able under certain circumstances to learn from the fundus of the eye whether hereditary syphilis is present in the organism.

185. Detachment of the Retina and Its Causes

The manifold forms and the varying extent of detachment of the retina require a quiet study of individual cases with the ophthalmoscope, and it is possible to indicate here only the most important characteristics. Our patient has the commonest form, the one associated with high myopia, of which it is the complication most to be dreaded, but it is also produced by other causes.

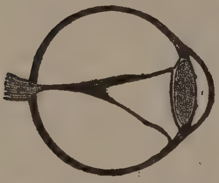


FIG. 135. — Detachment of the Retina.

I. When Should the Physician Suspect a Detachment of the Retina?

When a detachment of the retina is about to take place the patients very often complain of subjective sensations of light, the so called photopsias, flickering, fiery balls, flashes and sparks before the eyes. These symptoms are probably due to lacerations of the retina, and they may continue to exist long after the detachment has taken place.

Another symptom is metamorphopsia; the objects seen appear to be distorted, bent, or interrupted. This symptom is not necessarily indicative of a detachment, for the alteration of the rods and cones produced by chorioiditic patches in the macula may cause it, but when it is complained of, the possibility that it may be caused by a detachment must be borne in mind.

The color sense is very often impaired. The patients confound blue and green, or can no longer perceive blue. Probably this is due to phenomena of absorption in the yellow subretinal fluid. Hemeralopia may also be present; the vision is worse on cloudy days and in the evening than in the bright daylight.

The impairment of the vision is the most important symptom, the one that drives the patient to the physician, and in many cases it suffices to excite suspicion of the presence of a detachment. As the retina is extremely sensitive to nutritive disturbances it can be readily understood that after a short time the function of the detached parts is very greatly impaired and the central vision is much injured when the detachment is situated in the macula. Still more characteristic is the defect of the peripheral vision, the contraction of the visual field. The patients state that a dark cloud seems to lie in front of their eyes. As the favorite site of the detachment is in the lower half of the eye the patients may say that they can no longer see anything in the upper half of the field of vision.

II. The Objective Demonstration of Detachment of the Retina

Even the physician who has had no great amount of practice with the ophthalmoscope is able to recognize a detachment of the retina. All that is necessary is to illuminate the eye with the mirror and, while we watch the illuminated pupil through the aperture, to direct the patient to look several times in succession up, down, to the right, and to the left. This manœuvre is absolutely essential in order that we may illuminate the extreme periphery of the fundus. Under normal conditions all parts of the fundus return to us a clear, bright red reflex, but as we look into this patient's eye we see dark opacities float about in the vitreous as he looks in different directions. These opacities show that the nutrition of the vitreous is impaired and that the tissue is permeated with abnormal constituents, exudates and hemorrhages. As he looks downward and outward the red reflex suddenly becomes dull, partly gray, greenish gray, or in places whitish. When the subretinal effusion contains an admixture of the coloring matter of the blood the detachment inclines to be greenish. This phenomenon renders the suspicion that a detachment is present very strong; otherwise the reflex can assume such a greenish gray color only when dense preretinal opacities are present in the vitreous.

One peculiarity of the detached retina is that it is apt to lie in folds, so that the picture reminds us of the delineation of mountain ranges on the maps. We see white stripes with dark shadows between them; the white stripes correspond to the elevations, the shadows to the depressions of the folds of the retina.

The retinal vessels present three peculiarities. They describe large curves, are visible in some parts of their courses and disappear in others, which is readily explained by the folds. They appear to be very small, because the bulging of the detached retina reaches far forward into the space of the vitreous, so that the refraction in these places is hypermetropic and the vessels are accordingly seen magnified to a less degree. They also appear to be very dark or black, because they are seen partly by transmitted light. Some light always passes through the detached retina and is reflected back by the chorioid, so that the vessels appear to be dark just the same as opacities of the refractive media when they are seen by transmitted light.

A detachment of the retina can be recognized in this very simple way by throwing light into the eye by means of the mirror of the ophthalmoscope, but this must not be done by a hasty glance, we must investigate the eye carefully in all directions. In many cases it is easier to detect the detachment in this way than by the indirect method.

The next step is to ascertain the extent of the detachment and the condition of the papilla. Its margins may gradually flatten out into the normal retina, but sometimes the detachment is purse shaped and overhangs the papilla. If the exudate behind the retina is of a serous nature the reddish tone of the chorioid can still be seen through the gray, crumpled looking retina.

In many cases we are able to find a tear in the detached retina through which the fundus is clearly revealed, and we can see the intervascular spaces of the chorioid, or pathological changes in the vessels. These tears are usually in the periphery of the fundus, most often above. If, instead of serum, an effusion of blood, or a tumor, lies behind the detached retina the appearance is not clear, but dark brown or black. When the detachment is total we can perceive scarcely any red light in the wavelike, fluctuating gray of the retina.

By means of the direct method we can not only distinguish the individual characteristics of the detachment, but can also ascertain the difference between the refraction of the detached and of the still adherent portions of the retina. When the detachment is flat and very transparent it may happen that the only way in which its presence can be recognized is by this difference in refraction. In an emmetropic eye we find a high degree of hypermetropia on the surface of the detachment. If the eye is highly myopic the refraction of the surface of the detached retina may be a less degree of myopia, emmetropia, or hypermetropia.

To confirm the diagnosis we test the field of vision. This test in our patient reveals a large defect in the upper half, corresponding to the detachment of the retina in the lower part of the eyeball. We learn at the same time by this test what portion of the retina still retains its power of function.

III. Causes of Detachment of the Retina

The retina is but loosely adherent to the chorioid, its rods and cones only dip between the processes of the cells of the pigment epithelium, and it is firmly attached at only two places, at the papilla, where the fibres of the optic nerve pass over into it, and at the ora serrata. When a retina is totally detached it forms a funnel open in front and attached behind to the papilla, and has been aptly compared by *v. Arlt* to the flower of the convolvulus.

From the purely pathological standpoint there are two possible ways in which a detachment of the retina can originate. The retina may either be pressed out from the chorioid into the chamber of the vitreous, as by exudates from the chorioid, by hemorrhages, or by tumors of the chorioid and retina, or it may be drawn into the vitreous chamber by a shrinkage of the vitreous. But as these two ways of origin cannot always be distinguished clinically I prefer the clinical point of view from which to consider the etiology.

The first question in every case of detachment is whether it has been produced by fluid, or by a tumor, for on the answer to this question depends the fate of the eye and our therapeutic efforts. Sarcoma and glioma are the tumors principally met with in the eye, and when they are present not only the eye, but life itself is endangered and immediate enucleation is necessary, but an entirely different form of treatment is to be adopted when the detachment is due to an effusion of fluid. Even when a cysticercus is present between the retina and the chorioid, which rarely happens, we try to save the eye by the removal of the parasite, and in all other detachments caused by the effusion of fluid we try to save what we can of the vision. Hence it is of serious importance to determine which of these causes has produced the detachment.

When a severe hemorrhage from the chorioid produced by an injury has caused the retina to become detached, the history of the case will lead us to the correct diagnosis. Difficulty in the differential diagnosis is met with only in the detachments that occur spontaneously. First, we have the patient make little jerking movements of the eye while we are looking into it; if the retina floats back and forth the detachment is probably of a serous nature. The dark pigmented node of a commencing sarcoma can often be seen through the detached retina, which frequently has a speckled appearance over it. In the early stage of a tumor the nature of the detachment is also characteristic. There are no folds in the retina, as there are when it is spontaneous, but it protrudes as a smooth, unwrinkled prominence into the eye. This picture, however, can be seen only in a certain stage of the development of the growth. It is a well known clinical fact that fluid finally appears with all sarcomas of the chorioid and increases the detachment of the retina. Then we have a detachment caused by a tumor plus an effusion of fluid, and frequently the neoplasm can no longer be seen with the ophthalmoscope. Very small sarcomata of the chorioid may induce extensive detachments of the retina, and in such cases we have to rely on three methods of examination.

First we test the intraocular tension. This is distinctly minus in our patient, as it is very apt to be when the detachment is caused by fluid. A growing tumor on the contrary readily induces a condition of secondary glaucoma. The second method is that of transillumination with *Sachs'* lamp. The eye is anæsthetized with cocain, the point of the instrument is placed on the sclera and moved about from place to place, while we watch to see whether the light in the pupil remains red wherever the instrument is moved. The pupil constantly remains bright and red when the retina has been detached by a serous effusion, but when a sarcoma is present the dense tumor does not allow the light to shine through and the pupil is darkened. Extremely valuable as this method is it has its limitations, for dense hemorrhages may also obstruct the light. Finally, we may employ the method suggested by *Schmidt-Rimpler*, that of feeling of the sclera with a probe to detect a place where the increased resistance indicates it to be the site of a tumor.

Detachment of the retina may be due to very different causes. We meet with it for example in albuminuric retinitis and exudative chorioiditis. More often it occurs in senile eyes without having been preceded by any disease whatever. Contusions of the eye may produce it by causing hemorrhages or serous effusions. It may accompany the cicatricial contraction after perforating wounds with loss of vitreous and incarceration of the uvea. But the *spontaneous detachment met with in high myopia* is the most frequent form and should be the first to be thought of in every case.

The frequency of detachment of the retina in myopia depends very much on the degree of the nearsightedness. It is certain that the danger increases with the degree, and to give some idea of its frequency I will say that it occurs in about 5% of the persons in whom the myopia is over 10 D. Ordinarily this complication is unilateral, and we may comfort these unfortunates with the fact that the second eye is spared in over two thirds of the cases. The question has not yet been settled concerning the way in which this form of detachment of the retina takes place, but it is to be expected that further information in this regard will be obtained as our knowledge increases concerning the true nature of myopia. Various theories are held at the present time.

The theory of secretion and exudation was founded by *v. Arlt.* According to this the primary serous effusion comes from the chorioid and the pressure exerted by this exudate together with the detached retina causes a partial condensation of the vitreous and resorption of its fluid.

Stellwag v. Carion founded the theory of retraction. He thought that the detachment of the retina was produced by a contraction of the vitreous, the fibrils of which were diseased, and that the subretinal fluid was formed secondarily to fill the vacuum thus produced. This theory found a most zealous and distinguished advocate in *Th. Leber*, who supposed that the detachment of the retina was preceded by a chronic inflammation in the uveal tract with processes of contraction in the vitreous, which finally resulted in a laceration of the retina and the entrance of the intraocular fluid between it and the chorioid through the aperture thus formed. In fact *Nordenson* found on pathological examination a fibrillary degeneration of the vitreous, and also in some cases a rupture of the retina. If the retina remains united with the shrinking vitreous and no laceration takes place, the subretinal effusion must be produced by exudation, and thus far the theory of retraction cannot do without the assumption of a subretinal exudation of fluid.

To give some idea of the origin of this process of contraction *Leber* has published pictures to illustrate the genesis of spontaneous detachment of the retina, from which it appears that a cellular fibrous tissue is found with a certain degree of regularity on the inner surface of the retina and in the domain of the vitreous, which enters into the depths of the folds of the retina, fills them partially, or is stretched over their openings. *Deutschmann* also advanced the idea that a cicatricial process takes place in the ciliary body and that the detachment of the retina is to be ascribed to an equatorial chorioiditis which causes nutritive disturbances and a shrinkage of the vitreous. These lesions exert a tension forward upon the ora serrata and the retina. In opposition to this ophthalmoscopic theory of an equatorial chorioiditis *Leber* discloses a tension exerted upon the retina from the following pathological facts. Folds of the retina which are made adherent by such tissue are found in detachments. The retina may also be drawn inward at the place where the retinal funnel spreads out laterally by a membranous layer stretched transversely over the opening. The retina also often appears to be greatly stretched at the papilla and in its anterior portion. More than this it is shown that the pars ciliaris is involved in the detachment. *Leber* has found that layers of cells may proceed from the ciliary portion of the retina and grow over its inner surface. These layers of cells may extend not only over the outer surface of the vitreous and the inner surface of the retina, but also over the outer surface of the latter. These layers of cells can also form membranes resembling the hyaloid layer on the inner surface of the posterior part of the retina, and we can imagine that the contraction of these fibrocellular membranes on the inner surface of the retina may finally result in a detachment. The compressed vitreous must then undergo a fibrillary condensation. The opponents of this theory think that the changes in the vitreous are the actual results of the compression exerted upon it by the primary effusion of fluid together with the detached retina. It is very probable that the fibrillary condensation of the vitreous takes place in this way when the detachment is caused by a tumor, but *Leber* maintains in opposition to *Greef*, it seems to me correctly, that the condensation of the vitreous may be essentially different in a serous detachment. The condensed tissue of the vitreous adheres to the inner surface of the retina by predilection, while the most anterior portion of the retinal funnel, where one would expect to find the greatest condensation, may be occupied only by serous fluid.

In spite of these pathological facts, which make a traction on the retina probable, I do not consider it proven that the changes to be seen in the pathological preparations form the primary cause of a spontaneous detachment of the retina. As *Leber* himself says, it is necessary for us to infer from the changes the forces which have been active. It may be that these pathological changes, especially the formation and contraction of these fibrocellular layers, are the secondary results of other causes. It is not correct to urge against this theory that the occurrence of lacerations in the retina is comparatively rare. Even when the retina is not torn until the fluid has been forced forward to this place by an adhesion between the retina and the chorioid, conditions of retraction may be present on the inner surface of the retina and in the vitreous. It is also not right to say that according to *Leber's* theory the composition of the fluid in front of the retina must be the same as of that behind it. As long as no perforation of the retina takes place in the process of contraction and no fluid from the vitreous chamber can get behind it, the subretinal fluid must be formed by exudation. It may occasionally contain such substances as cholesterin, which are not found in the vitreous chamber. On the other hand it is difficult to explain by *Leber's* theory the clinical fact that such a spontaneous detachment may spontaneously reattach itself, sometimes within a remarkably short time. *Leber's* explanation, that the same forces that produced the detachment later bring about the reattachment by acting more on the outer side of the retina, is worse than forced, for if contracting cords in the vitreous have detached the retina their tendency to contract will increase as soon as the resistance has yielded. I believe that the forces which bring about detachment of the retina are not so slight as they have been supposed to be. While therefore I adhere on the whole to the retraction theory I have to admit that beyond a doubt there are many cases in which the cause is a primary effusion behind the retina. It is interesting in this connection that *Wessely* has been able to produce experimentally, by the local application of steam to the sclera, detachments of the retina which were caused by such effusions.

IV. Treatment and Prognosis of Detachment of the Retina

The treatment of detachment of the retina is one of the most thankless and unsatisfactory duties we have to perform in ophthalmology. In order to obtain a proper estimate of the value of our therapeutic efforts we must take into account that it may recover of itself. According to *Uhthoff* a spontaneous reapposition and recovery of a detached retina takes place in about 8% of the cases. The place where a detachment has formerly existed can be recognized from the chorioiditic changes that are present in the form of bright white, or yellowish white cords with pigmented margins. A pigmented band can frequently be seen to run along the margin of a former effusion of fluid. It also happens that a partial detachment may remain stationary. I know an officer who has had a detachment of the retina in each eye for years, and is still able to attend to his duties. But in the great majority of cases a detachment of the retina tends to increase, or to become total. A symptom that may be observed regularly is that the detachment changes its position. In many cases the effusion begins in the upper part of the eyeball and then sinks, while the retina is replaced above. An apparent recovery may take place when the detachment was at first in the region of the macula and then sinks below it; the macula may regain a part of its functional power in such a case, while a slight peripheral contraction of the field of vision causes little trouble. It is due to this change in the extent and position of the detachment that fluctuations in vision and temporary subjective improvements often appear. The behavior of the detached retina as regards its function varies a great deal. Sometimes its functional power is destroyed in a very short time, while in other cases it is partially preserved. The eye usually undergoes other changes when the detachment becomes total. The intraocular tension gradually decreases in consequence of the shrinkage of the vitreous, and the eye becomes soft. Sometimes inflammatory symptoms appear in the iris and we find synechiæ or atrophy. The lens finally suffers from complicated cataract, due to disturbances in its nutrition. Attention is called to such a cataract by the absence of projection and a yellowish appearance of the lens in which calcification may take place. As the lens continues to shrink it becomes tremulous, and luxations in consequence of the atrophy of the zonule are not uncommon. Finally the eye becomes totally amaurotic. We cannot see the detachment in such an eye with cataract, but we can be certain of its presence when the tension is very low and the projection of light is bad.

We have at our disposal a conservative and an operative method of treatment. I agree with many other ophthalmologists that the conservative method should be tried first. This consists of rest in bed, sweating, pressure bandage, and subconjunctival injections of salt solution. If this treatment proves ineffectual we may evacuate the subretinal fluid in the following manner. After the eye has been cocainized we make an incision through the conjunctiva bulbi, undermine it and make an incision through the sclera and chorioid as far back as possible between the muscles over the site of the detachment. A large quantity of yellowish, serous exudate flows out through the opening. We can now perforate the sclera in one or more places near the incision with a fine galvanocautery in the hope that the aseptic inflammation thus induced will lead to an adhesion and later to a firm union between the retina and the chorioid. An infection is not to be feared, as these wounds lie beneath the conjunctiva, the opening in which is finally closed with sutures. Division of the bands in the vitreous and injection of the vitreous of rabbits have been recommended, in addition to puncture and cauterization, but I do not use these measures.

186. Glioma and Pseudoglioma

The mother of this little child, 3 years old, discovered a few days ago a yellow color in one of his eyes, and she is convinced that the eye is blind. Two things only are possible: this is either a case of glioma, and if so the diseased eye must be enucleated as quickly as possible in order to save the life of the child, for glioma is one of the most malignant of tumors, or it may be one of pseudoglioma, when we leave the eye alone.

I. Nature and Course of Glioma

Glioma is a malignant neoplasm that starts from the retina. It is one of the rarer diseases of the eye, met with in most eye clinics in the proportion of one case in several thousands of patients.

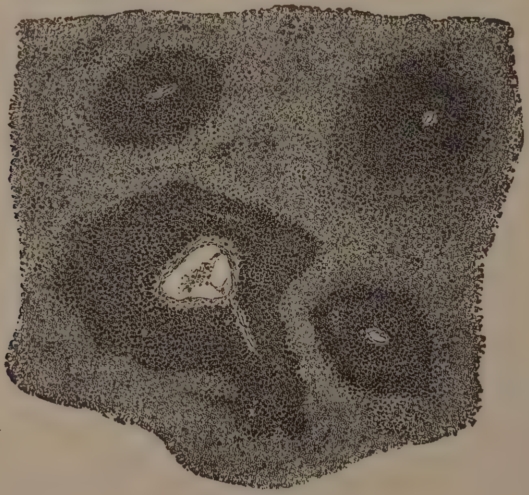


FIG. 136.—Glioma

The first clinical characteristic of the tumor is that it is met with exclusively in early life. Occasionally it has been observed as late as the twelfth year, but ordinarily it occurs during the first four years. According to a compilation made by *Wintersteiner* two thirds of all the children have not completed their third year. This indicates a congenital predisposition to this form of tumor, which is also suggested by its second clinical characteristic, its appearance in families. Quite a number of observations have been made in which it occurred in a large number of the members of one family; for example, *Lerche* reported the deaths of four out of seven brothers and sisters from glioma. We may therefore imagine that it is a congenital disease, the more so as it has been observed to be present at birth, but the rudiment is often very slight at that time and the tumor develops later.

Glioma is usually present in only one eye, yet the proportion in which both are affected, almost 20%, is by no means small. It must not be thought that the tumor has passed from one eye to the other when both are involved, the disease is primary in both retinæ, even though one eye is attacked after the other.

The Various Stages in the Clinical Course of a Glioma

Glioma passes through several stages, the same as intraocular sarcoma, though they are not sharply defined.

First Stage: The Amaurotic Cat's Eye

As the disease occurs in little children the beginning of the growth can be observed only in exceptional cases, for instance, in those in which a small tumor is detected in the second eye during the examination. The little patients make no complaint of their visual disturbances, so the first that is known, as a rule, of the existence of a glioma is when the mother sees it in the eye of her child. During this first stage a bright yellow color shines from within the eye, producing a clinical picture that was named by *Beer* amaurotic cat's eye, because the eye is blind and shines like the eye of a cat. This reflex is usually yellowish or reddish yellow when the particles of the tumor are visible, or rather bluish white when the reflex comes from the detached portion of the retina.

The second symptom is the blindness. As a rule, vision has been totally lost before the tumor is discovered. Of course vision must still be present when the tumor begins to develop and the disturbance of the vision must depend on the part of the retina from which the tumor originates, whether the macula or the periphery is the part first affected, and on the degree to which the retina is detached, but all this is of no practical importance. The only thing we can do is to test the reaction of the pupil and to notice whether the child looks at a light or not.

The pupil in this case is fixed and dilated, but dilatation of the pupil does not generally appear until the increase of the tension has begun and the disease has entered its second stage. The opportunity is scarcely ever offered to examine with the ophthalmoscope the very first stages of this disease. We may see in this case an extensive detachment of the retina from which protrudes a reddish yellow knob into the vitreous, which is bounded by distinct furrows and has a rough surface. The color is not uniformly yellowish everywhere, but lustrous white spots are to be seen in places, which are patches of disintegration. Near it are little hemorrhages, and the surface of the eminence is covered with vessels. In other cases the masses of the tumor may be crumbled away at an early period, when the vitreous will be filled with little roundish spots and nodules.

Second Stage: Stadium Glaucomatosum

The tumor continues to grow and the disease enters upon its second stage, that of secondary glaucoma. The iris and the lens are driven forward more and more, the iris becomes atrophic, the pupil dilated, and the sinus of the anterior chamber obliterated. The anterior chamber is then shallow, the cornea loses its sensibility and is often opaque. The anterior ciliary vessels are abnormally distended and tortuous because of the increased tension within the eye; we have in short the typical picture of glaucoma. In this stage the little patients begin to suffer from pain and to refuse their food. If the process goes on still further the lens gradually becomes opaque, making the yellow appearance of the eye more indistinct. Sometimes this formation of cataract appears very early, or the lens may be luxated into the anterior chamber. If the intraocular tension is tested in this stage it will be found to be distinctly increased, but not very greatly, in spite of the habitus glaucomatosus, because the capsule of the eyeball in the child is soft, extensible, and yields to the pressure.

Third Stage: Ectasia Bulbi and Exophthalmos

Next the form of the eye changes and the disease passes into another clinical stage. The anterior zone of the sclera begins to give way, becomes broader and assumes a blue gray color, because the pigment of the uvea shows through. The circumference of the cornea increases, producing the condition known as megalocornea. An anterior scleral staphyloma is formed in this way, while behind the insertions of the muscles the capsule of the globe stretches in the same way and the eyeball is enlarged at the posterior pole. The cornea, which has already become opaque, may readily become perforated and then a part of the tumor escapes.

Even without this last complication the tumor finds its way outward altogether too soon. It may grow along the vortex veins, or the anterior ciliary vessels, perforate the sclera and give rise to episcleral nodes. Or the perforation may take place behind, through the places of entrance of the posterior ciliary vessels, or more likely along the optic nerve and its sheath into the orbit. Then exophthalmos is added to the ectasia of the globe.

Wherever the perforation may take place two symptoms are always to be met with. The pain ameliorates somewhat with the reduction of the intraocular tension, and the masses of tumor, freed from the pressure that has impeded their proliferation, increase with a rapidity that is often remarkable and the disease enters its final stage.

Fourth Stage: Extraocular Proliferation and Metastases

If the perforation has taken place in the anterior segment of the globe large tumors are quickly formed, which often reach the size of a fist, or may be even larger. The orbit is distended, the preauricular glands are enlarged, the parotid region is swollen, and the swelling spreads over the angle of the jaw. Then come metastases in the brow, the scalp, and the roof of the skull. The rest of the course does not need to be delineated. Often we are first able to diagnose the commonest metastases, those within the skull, from the functional disturbances of the central nervous system to which they give rise. Clinical experience has taught us that when a protrusion of the eyeball occurs the infiltration along the optic nerve has already reached the cranial cavity. The principal symptoms are convulsions and paralyses, and death finally takes place in unconsciousness and coma. If the course of the disease is prolonged symptoms may appear in the other eye. It may become blind because the tumor has reached the base of the brain along the optic nerve and compressed or involved the chiasm. A mydriasis calls our attention to this amaurosis. The average duration of this disease till death is about one and a half years.

A glioma starts from the granular layers of the retina and consists, on ordinary examination, of densely packed roundish cells that resemble the cells of the granular layers, having large nuclei and narrow edges of protoplasm. If *Golgi's* method of staining is employed, the oval cells are seen to have numerous fine long offshoots that radiate in all directions. These are glia cells. Together with them we find ganglion cells.

A second characteristic of glioma is that the tumors are extremely rich in blood vessels, and that the tumor cells frequently seem to be arranged like rosettes about the vessels, so as to resemble, to a superficial glance, tubular glands, but the farther we go from such a vessel with its mantle of cells the worse do the tumor cells take the stain. This is because the cells which are situated the more peripherally fail to receive nutrition and die. Thus proliferation and necrosis go hand in hand. Places that have undergone fatty degeneration may be seen lying here and there between the masses of dead cells. We also frequently meet with hemorrhages and other signs of regressive metamorphosis, such as calcification and hydropic degeneration of the cells. *Hirschberg* differentiates two varieties, glioma endophytum and glioma exophytum. In the more uncommon form, glioma endophytum, miliary nodules are scattered about in the cavity of the vitreous from which growth takes place until the eyeball has been filled. In glioma exophytum the growth of the tumor nodules proceeds toward the chorioid after the retina has been detached. The nodes lie at first upon the pigment epithelium, which soon shows signs of proliferation and finally atrophies. The tumor then presses through the elastic lamina into the chorioid where the masses can spread out superficially and may proliferate into the anterior chamber, or vessels may grow out from the tumor into the chorioid and be accompanied by the tumor cells. The growth may also proliferate into the chorioid from the papilla.

It is self evident that in dealing with so malignant a growth the only treatment can be a timely enucleation. The prognosis on the whole is very bad. Nine out of ten cases that came under the observation of *Vossius* died. The shortest interval between the operation and death was two months, the longest five and a half years. According to *Hirschberg* the percentage of cures is only 6.5%, according to *Wintersteiner*, 13.7%. From three to five years must elapse before we can be certain that there is no danger of metastasis or of disease in the other eye. We can calculate on a permanent cure only when the optic nerve is still intact and the tumor is confined to the retina. The cases are frightful in which we discover a glioma in both eyes. If the tumor has already perforated the eyeball we must exenterate the orbit, removing the periosteum; in this way we may at least prolong life and mitigate the suffering of the child, but we cannot prevent metastases, and when we are called upon to operate on a recurrence we know that the disease is incurable.

II. Pseudoglioma

Sometimes it is very difficult to tell when a glioma is really present. This is the case when cicatrices in the cornea, or a corneal staphyloma produced by an ophthalmia neonatorum, obstruct the view into the eye, or when the glioma has caused a cataract to develop early, or when a hemorrhage into the anterior chamber renders an ophthalmoscopic examination impossible. A simple detachment of the retina may also render the diagnosis difficult, for such a detachment is not uncommon in children, and when the glioma is still small and covered by an extensive detachment of the retina its recognition may be delayed until it is too late to prevent its extension. In general a retina that has been detached by a serous effusion floats about, while one that has been pushed off by a tumor does not do so; but exceptions to this rule are met with. The intraocular tension is usually reduced in simple detachment and increased when a tumor is present, but it may be normal when the latter begins to develop.

It is not such a serious error to mistake a glioma for a sarcoma, for in either case the eye must be enucleated at once, but such an error is not likely to be made, as sarcoma occurs very rarely in children.

Advanced tubercles of the chorioid, which have filled the cavity of the vitreous with granulation tissue and have led to secondary glaucoma with ectasia of the sclera, have been mistaken for glioma. Tuberculosis has been found when the diagnosis was that of glioma, and vice versa. A cysticercus can sometimes produce a condition which closely resembles that caused by glioma, but this parasite is very rarely met with in children.

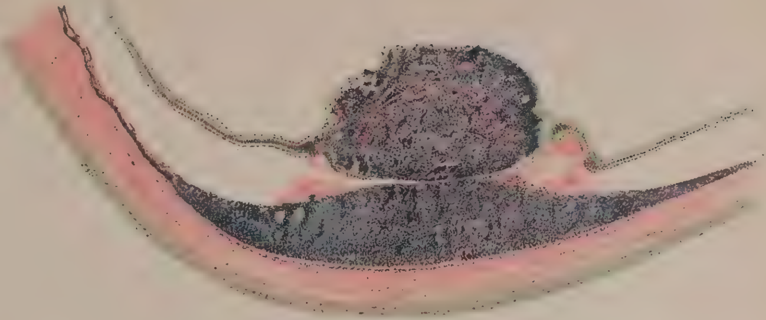
In practice we much more often meet with the condition known as *pseudoglioma.* We understand by this term chronic inflammations of the chorioid and ciliary body which have led to a large fibrinous exudation in the vitreous. When in the course of time such exudates organize they may give a golden yellow reflex from the depths of the eye in a way quite analogous to that seen in glioma. Other diseases that may give rise to the clinical picture of the amaurotic cat's eye are abscesses of the vitreous following injuries, and the benign form of metastatic uveitis from meningitis or other infectious diseases. Hemorrhages into the vitreous that have become organized and have caused detachment of the retina can produce the same picture, in a certain stage.

Sometimes it is very difficult to differentiate these changes in the vitreous from glioma. In many cases the history gives us important data for the diagnosis. We investigate first whether the eye has received an injury which may have led to an abscess of the vitreous with subsequent organization into connective tissue, and see if we can find any scars. Nothing of this sort appearing we ask whether the child has been suffering from any sickness characterized by fever, rigidity of the neck, convulsions, or other cerebral symptoms, which may have occasioned a benign metastatic uveitis. Such a metastatic uveitis sometimes appears after an attack of disease that clinically ran a relatively benign course.

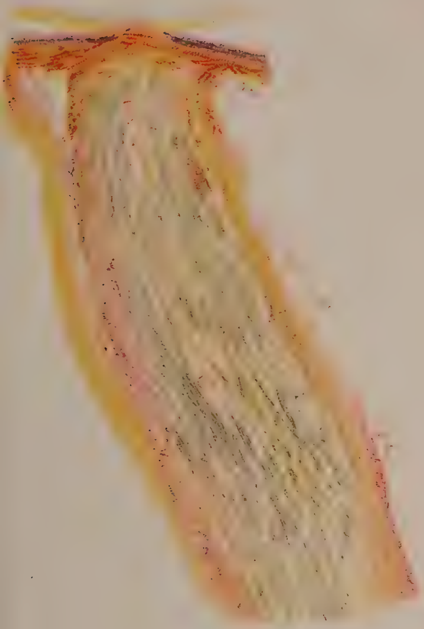
But we cannot depend on the history alone when it furnishes us no such data, we must rely on the results of the examination of the eye itself. The first point of differentiation between glioma and pseudoglioma is the color of the reflecting mass in the vitreous, which is usually a reddish yellow in the former and rather of a metallic brassy yellow in the latter, but this is not a very positive sign.

A point that is of more importance is the presence or absence of vessels. Vessels can be seen, almost as a rule, by means of focal illumination in glioma, while in pseudoglioma the exudates are frequently destitute of them. Still quite a number of cases of glioma have been reported in which no vessels were visible with the ophthalmoscope, and on the other hand an exudate in the vitreous that is undergoing organization may be very well provided with them.

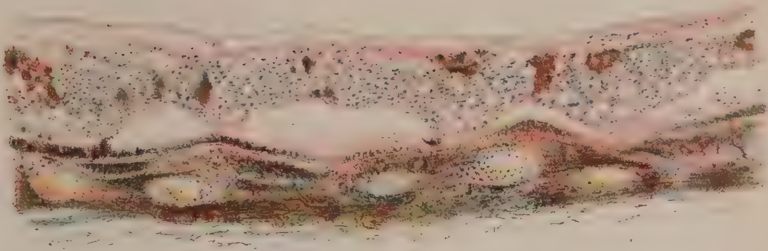
A third point is that the surface of a glioma is usually distinctly rough, while, as a rule, that of an exudate is smooth, but this condition also is not always diagnostic. If a glioma is pressing against the lens its surface may be smooth, while an exudate that is undergoing contraction may be nodulated.



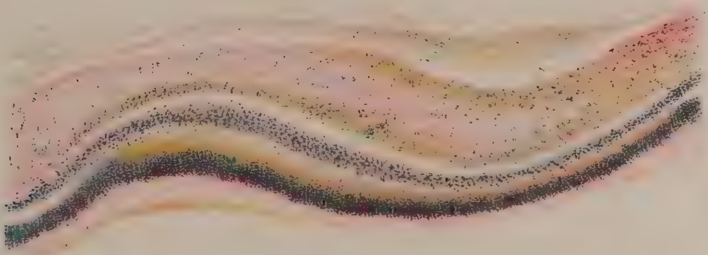
b Sarcoma of the choroid or vascular tunic (Diagnosed ophthalmoscopically).



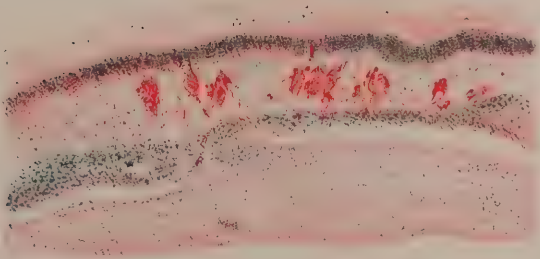
a Atrophy of Optic Nerve in Tabes.



c Pigmentary degeneration of the retina.



d Retinitis leukaemica.



e Retinitis albuminurica.

Rebman Company, New York.

The surface of a glioma that is growing inward may also appear as much like jelly as that of an exudate.

The fourth point is a very important one, whether any signs of a past iridocyclitis can be found in the eye. We look for posterior synechiæ at the margin of the pupil, we examine carefully the posterior surface of the cornea for traces of deposits on *Descemet's* membrane, or misplaced pigment. The pupils are frequently dilated in glioma and are contracted in iridocyclitis.

The fifth point is the form of the eyeball, which is often diminished in size in pseudoglioma, but is rather enlarged by a growing tumor, because the capsule of the eyeball is very capable of being stretched in children.

Finally a sixth valuable point of differentiation is furnished by the intraocular tension. The tension of the eye is generally lowered when exudates in the vitreous have brought about the picture of a pseudoglioma, while in glioma it is either normal or increased. In this case all of the indications point to glioma, and there is no doubt in regard to the diagnosis. We must advise the mother to have the eye of the child enucleated.

Two Months Later

A recurrence appeared a few weeks after the enucleation, some idea of which can be obtained from the accompanying illustration. Exenteratio orbitæ was performed, but after a few days it was evident that the tumor had extended to the brain and finally the child died. To round out our conception of this disease I will give the findings at the autopsy.

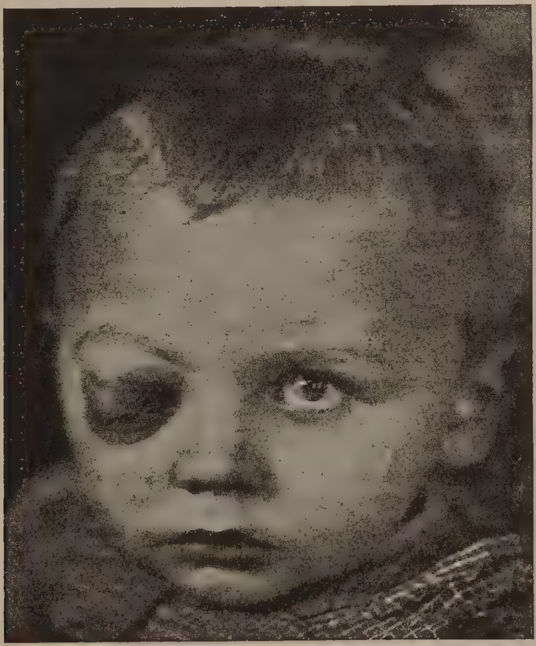


FIG. 137.—Recurrent Glioma.

We found on the right side of the brain along the sagittal fissure a brownish yellow discoloration of the pia mater which was due to an old hemorrhage. Over the lateral portion of the cerebral cortex on each side there lay scattered about in the pia, distinctly following the vessels, numerous little irregular glassy white nodules that varied in size from that of the head of a pin to that of a hemp seed. These gradually disappeared toward the top of the convexity. They were most numerous on the right side, where they could be followed along the vessels into the fossa of *Sylvius.* At the base of the brain, where the olfactory bulb and the chiasm are normally visible, we found resting on the brain a fungous mass of irregular, fissured form, and of a bloody, brown red color, about as large as a small apple. This mass extended backward into the infundibulum to the right of the apex of the temporal lobe, over part of which it passed, and ended at the level of the top of the olfactory bulb, of which we could find no trace. The tumor was soft and could not be removed without loss of substance from the brain. The entire pia at this place exhibited just the same glassy white roundish nodules as those that were found on the convexity, except that they were larger. The pia of the cerebellum was thickened and in it also were scattered about little nodules of the tumor.

XIX

Functional Testing of the Eye

187. The Control of Disturbances in the Functions of the Eye by Means of Focal Illumination, Transmission of Light through the Refractive Media and the Examination of the Fundus

When a patient complains of a disturbance in the function of the eyes the history should be taken and an objective examination made before we proceed with the functional test, because the only person who is able to judge correctly concerning the functions of the eye, and their disturbances, is the one who can compare the statements made by the patient in the test with the objective condition of the eye. The methods employed in this objective examination are focal illumination, transmission of light through the refractive media and the investigation of the fundus.

I. Focal Illumination

We begin with focal illumination because a functional disturbance may be due to a fault in the anterior segment of the eyeball. The light from a lamp, placed at the side of and a little in front of the patient, is concentrated with a strong convex lens upon the cornea in such a way that the apex of the cone of light falls upon the part to be examined. The place undergoing examination is then at the focus of the lens, and this fact has given the name focal illumination to the method. As the tissue in the immediate vicinity is in the dark, pathological changes can be seen very distinctly in the brightly illuminated area. The only precaution that needs to be taken is not to have the lamp placed too near the eye, so that the rays of light can be really brought to a focus by the convex lens. We are able to satisfy ourselves in this way concerning the condition of the cornea, anterior chamber, iris, the pupillary reaction, and, to a certain extent, of the lens, and even of the anterior portion of the vitreous.

II. Transmission of Light through the Refractive Media

The lamp is now placed at the side of and behind the head of the patient, and its light is thrown into his eye with the mirror of the ophthalmoscope, which is held firmly against the upper margin of the orbit of the examiner in such a way that he can look through the aperture. The pupil of the normal eye appears red when looked at in this manner, while opacities in the refractive media stand out as dark spots against the red of the pupil, and we can easily ascertain whether they are in the lens or the vitreous in any given case, by having the patient make certain movements of his eye; an opacity in the lens moves only with the eye itself, while opacities in the vitreous continue to float about when the eye is at rest.

We can also determine the position of an opacity by means of its *parallactic displacement with reference to the margin of the pupil.* For example, if an opacity is seen by transmitted light in the red pupil while the eye undergoing examination is looking straight ahead, it may be situated in the central part of the cornea, at the anterior pole of the lens, or still farther back; then if the patient is told to look up the opacity will seem to move toward the upper margin of the pupil if it is on the cornea, it will not seem to change its position if it is at the anterior pole of the lens, and will appear to move downward toward the lower margin of the pupil as the eye moves upward if it is at the posterior pole of the lens, because it lies behind the point of rotation of the eye.

Why does the pupil appear to be black under normal conditions? Why can we not see into it with the naked eye? Why does it appear to be illuminated only when we throw light into the eye with a mirror? These problems were solved by *Helmholtz.* The human eye may be aptly compared with a camera obscura. An inverted small image of the objects in the outer world is thrown upon the retina, just as upon the sensitive plate of a camera, but instead of a single lens the human eye has a series of refractive media. We cannot see into a photographic camera when we stand in front of it any more than into an eye, its aperture looks just as black as the pupil, because of the following law of physics. If we place a light in front of a camera at a distance of 2 metres, a sharply defined image of it is formed on the ground glass plate. If we darken the room, put the light in the place of the ground glass plate and the latter in the place of the light we again obtain a sharply defined image. *This shows that the rays of light which are sent out by the flame and are focussed into a sharply defined image on the ground glass plate return again to the flame. The image and the object stand in reciprocal relations to each other, so that we can have the two exchange places without any alteration of the distance between them. This also tells us at once that we can see into the eye of the patient only when we arrange matters so that the rays of light will proceed as though from our own eye, enter that of the patient, and then return to our own.* When we use the ophthalmoscope and throw light with it into an eye the rays return and, while part of them fall upon the mirror, some of them pass through the aperture into the eye of the observer. This is the principle of the ophthalmoscope.

III. Examination with the Inverted Image

After we have investigated the refractive media for the presence of any opacities which may have been the cause of the functional disturbance we examine the fundus to learn if any pathological changes are present. To do this we make use first of the so called inverted image (the indirect method). We throw light into the eye with a concave mirror, which gives a strong illumination, intercept the rays returning from the eye with a convex lens of the strength of 13 dioptres, which focusses them into an inverted real image that we examine through the aperture of the ophthalmoscope. The source of light is placed to the side of and a little behind the head of the patient, the examiner sits in front of him, places the ophthalmoscope against the upper margin of his own orbit, so that he can see through the aperture, and throws the light into the eye of the patient by means of the mirror. We usually examine first the optic nerve, and as this enters the eyeball on the nasal side of the posterior pole we direct the patient to look somewhat inward. The best way is to have him look at a distance past the examiner's right ear when the right eye is to be examined, or past the left ear when we wish to investigate the left eye. When the light is thrown into the eye in this position we see that the returning light has a brighter tone of color, which is due to the fact that it is reflected from the papilla, and this under normal conditions is colored more brightly, at least on its temporal side, than the rest of the red fundus, and we know that the optic nerve is directly in front of us. Now we hold the convex lens in a plane parallel to that of the face, about 5 to 7 cm in front of the eye of the patient, steadying it by placing the little finger of the hand holding it on his brow or temple, at the same time holding the arm in such a way that it does not obscure the source of light. These prerequisites for the examination of the fundus having been complied with it only remains for the student to learn by practice to focus his own eyes upon the image in front of the loupe by means of his accommodation.

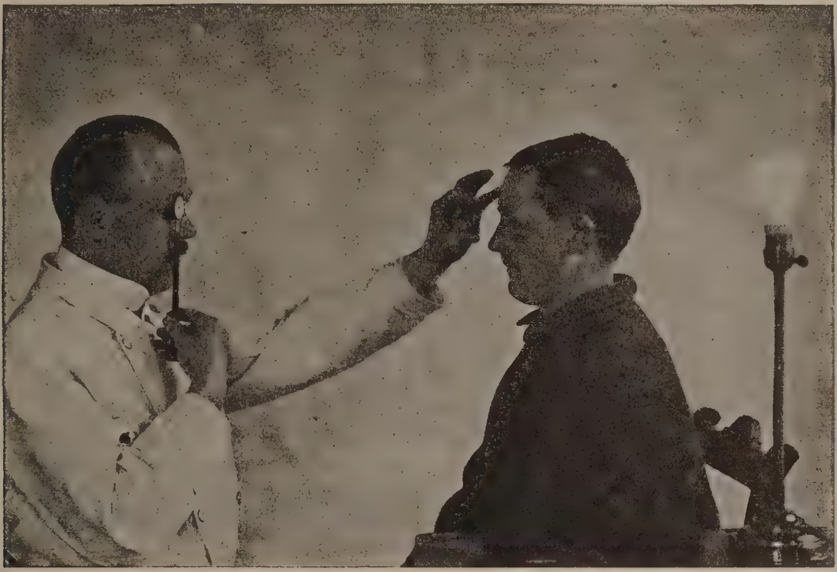


FIG. 138.—Examination with the Inverted Image.

The fundus is seen in the inverted image magnified only about four times on the average, but this method presents the advantage that by means of it we obtain at once a general survey over a large field. It is therefore to be used whenever we wish to make a rapid observation of the condition of the fundus.

IV. Examination with the Upright Image

The upright image, or direct method, is used when we wish to study more closely the details of the pathological changes in the fundus. If the eye being examined is normal in construction, the rays of light that return from within it are parallel, and if the eye of the observer is likewise emmetropic these parallel rays are focussed at once on his retina and he is able to see the fundus distinctly without any other aid, provided only that he brings his eye as closely as possible to that of the patient. We first throw the light into the eye with the mirror and then draw closer and closer to it without losing sight of the illumination in the pupil. The only bit of skill required is that the examiner learn to relax his accommodation, to look as it were at a distance. The picture of the fundus then appears entirely of its own accord. The field of view which we can command is quite small, but the advantage of this method is that what we see is magnified fourteen times. The further details of this method can be learned only from actual practice.

We also use, in testing the functions of the eye, methods by means of which to determine its refraction, as well as perimetry and tonometry. The exactitude of ophthalmology depends upon the exactness of these methods of examination.

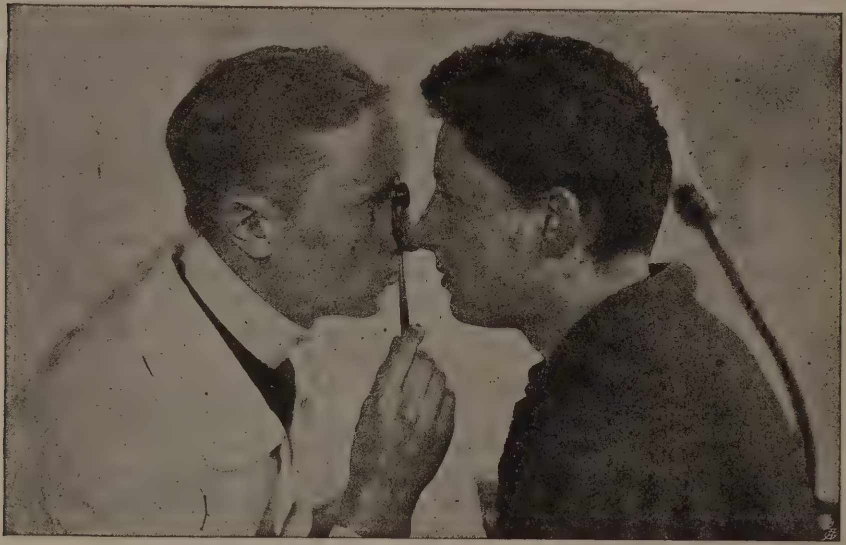


FIG. 139.—Upright Image.

188. The Numbering and Measurement of Spectacle Glasses

We have in our test cases two kinds of glasses, or lenses: convex and concave. We will take first a convex glass and show its optical action by the following experiment. Standing a few metres away from the window I hold the convex lens parallel to it and then bring a sheet of paper closer and closer to the lens on the side away from the window until we can see a small inverted image of the latter upon it, which is perfectly clear when the paper is at a certain distance from the lens.

How Is This Image Produced?

The physical *law of refraction* is that a ray of light is bent toward the perpendicular to the surface at the point of incidence when it passes from an optically thinner to an optically denser medium. A ray that falls on the spherically curved surface of a lens is refracted toward the perpendicular at its point of incidence, which in this case is a prolongation of the radius. The ray which is itself perpendicular to the refracting surface is the only one that passes through without being bent. The point through which rays of light pass without being deviated is known as the *optical centre of the lens.* This optical centre is situated at the actual centre when the lens is symmetrically ground; in unsymmetrical lenses it is situated nearer to the surface that has the greater curvature. All of the other rays which are parallel to the one which is vertical to the spherical surface are deviated toward the perpendicular at the point of incidence and converge to a point which is called the *principal focus of the lens.* The principal focus is therefore the point at which rays of light coming from infinite distance, and therefore parallel, that fall upon the lens are caused to unite.

From this drawing we see that the principal focus of a convex lens lies on the side opposite to that upon which the rays of light fall. The convex lens renders parallel rays convergent, it "collects" them into the principal focus and is therefore called a collecting lens.

Inversely, rays of light starting from the principal focus and impinging on the lens emerge from the latter in a parallel direction. Still more is to be learned from this experiment. Parallel rays are refracted so strongly by the lens that they unite on its opposite side. The picture of the window is sharply defined when the sheet of paper is exactly at a certain distance from the lens—in other words, the focal point of any convex lens is at a certain exact distance from its optical centre, and we call this the *principal focal distance.*

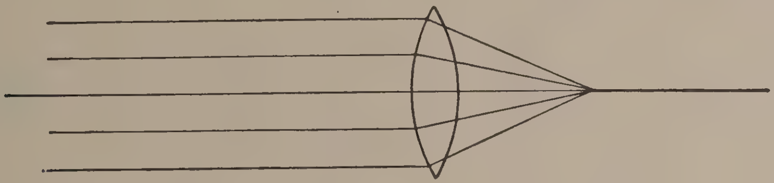


FIG. 140.—Union of Parallel Rays, Effected by a Convex Lens.

The length of the principal focal distance varies in different lenses and furnishes us with a measure of the refracting power, or strength of a lens. If we place in conjunction with the first convex lens another ground in the same way we find the distance at which the sharply defined image of the window is produced to be very much less than when either is used alone; the rays of light are evidently refracted much more strongly and the principal focal distance is shorter. From this we obtain the law that the shorter the principal focal distance of a lens the stronger is its refractive power, or, to express it otherwise, the refractive power of a lens is inversely proportional to its principal focal distance. Instead of placing two glasses of equal strength upon each other we naturally require the optician to grind a single lens of twice the strength.

The numbering of spectacle glasses is based on the principal focal distance, and it is necessary that we should have a *unit lens* to which we can refer in order to compare the strength of different glasses. The lens that is now generally accepted as the unit has a principal focal distance of one metre. Parallel rays of light that fall upon one side of such a convex lens are so refracted that they unite on its opposite side at the distance of one metre from its optical centre.

The refractive power of such a lens with a principal focal distance of one metre we call a *dioptre.* If we place two such lenses one upon the other the refractive power of the optical system is doubled and the parallel rays unite at the distance of half a metre, at 100/2 cm or 50 cm. Instead of placing two such lenses upon each other we may have a single one ground with a principal focal distance of 50 cm, and then we have a lens of 2 dioptres. A convex lens of 4 dioptres of refractive power causes parallel rays to unite at a distance of $100/4=25$ cm and so on. A lens of x dioptres has a principal focal distance of $100/x$ cm. Inversely, if the length of the principal focal distance of a lens is known, its refractive power can be given at once. A lens with a principal focal distance of 10 cm has 10 dioptres of refractive power. Hence when we wish to determine the number of a lens all we have to do is to ascertain its principal focal distance. But how is this to be done?

Let us take this glass which is worn by a patient who has been operated on for cataract. All that is necessary is to focus the image of the window upon the paper and to measure the distance from the paper to the lens. The distance is about 10 cm, and as the principal focal distance of this glass is 10 cm its strength is 10 dioptres. The focal distance of strong convex lenses may be measured in this manner by the direct determination of the focal point, but in practice this method is applicable only to strong ones. An attempt to obtain a picture of the window with a weak convex lens in this way will not succeed; a darkened room and a complicated preparation for the experiment are necessary for its success. We therefore use another method to ascertain the principal focal distance, but before we can deal with it we must turn our attention to another set of lenses, the concave glasses.

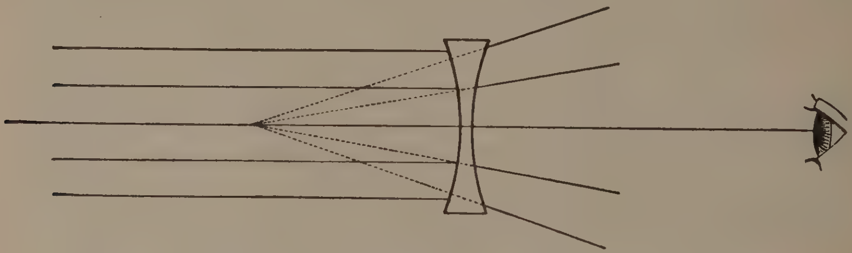


FIG. 141.—Dispersion of Parallel Rays of Light by a Concave Lens.

Concave glasses act in exactly the opposite way to the convex. They do not collect, but disperse the rays of light that fall upon them. The course of the rays of light through a concave lens is as follows:

The principal focal distance lies on the same side of a concave lens as the parallel rays that impinge upon it; it is therefore a *negative principal focal distance* and is indicated by the sign —. These two kinds of glasses act as forces in opposite directions and their focal distances serve as standards with opposite signs. This provides a very simple way in which to ascertain the strength of a lens; all we have to do is to neutralize it with a glass of the same strength ground in the opposite way, for if we place two such glasses together the effect of each is neutralized and a plane glass is formed. This is the so called *compensation* or *neutralization* method. The number of the glass that neutralizes the optical effect of the one being examined gives us the number of the latter.

One thing more is necessary—how to learn at once whether a given glass is convex or concave. This may be told by feeling of them when the lenses are strong, but not when they are weak. Another method, which likewise is applicable only to strong lenses, is to look through them at the second hand of a watch; the hand will look larger through a convex lens and smaller through a concave. A method which is applicable to all glasses and reveals even the minutest degrees, is that of *prismatic displacement*.

We may conceive of a convex glass as composed of two prisms with their bases together and their refracting edges directed upward and downward. If we hold a weak convex lens close in front of the eye and look through its centre, as it were directly through the dividing line of the two prisms, we see the lamp in front of us in its right place, but as soon as we move it slightly up or down the lamp appears to move in the opposite direction. If the glass is moved down the lamp seems to go up. This is explained by the prismatic action of a convex glass. A prism deviates parallel rays toward its base, so if such a double prism is moved downward from the line of fixation the rays that pass through the line dividing the prisms no longer enter the eye, but rays enter which have fallen upon the prism above and have been deviated downward, so we see the object along a prolongation of the line of entrance of these rays, i.e., above.

The prismatic action of concave lenses is the exact reverse. We may imagine a concave glass to consist of two prisms with their apices together. If we look at a distant object through a concave glass and move the latter before the eye the object makes an apparent movement in the same direction. Thus we are able to tell at once whether a patient is wearing a convex or a concave glass. We then take systematically from our test case glasses ground in the opposite way, beginning with the weaker numbers, 1 D, 2 D, etc., place them upon the glass to be examined, move the combination about before our eyes, and replace the trial lens with a stronger one until we obtain a combination with which we can see no apparent movement of an object looked at through it. Then we have learned the number of the glass from that of the trial lens needed to neutralize it. This method is very accurate, and variations of 0.5 and .25 D can be perceived at once.

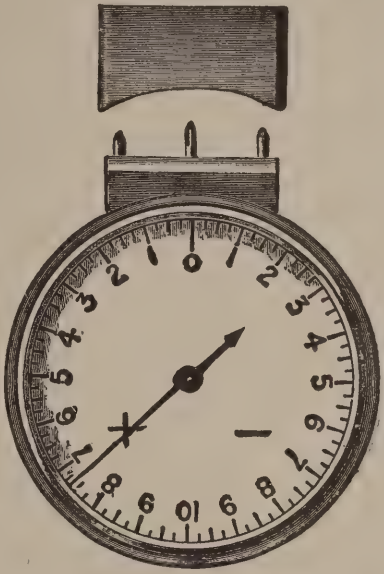


FIG. 142.—Lens Measure.

A very useful instrument for the same purpose is the *lens measure*. We press the spring against the centre of the glass and the indicator shows at once the refractive power of the surface in dioptres.

189. The Reduced Eye and the Visual Acuity

When a patient comes to us to have his eyes examined the first question for us to investigate is whether the eyes are *optically* normal or not, for abnormalities in the dioptric apparatus are very common causes of visual trouble. A human eye is normal when both its construction and its functional power are normal.

I have already compared the human eye to a camera obscura and the comparison may be extended. As the photographic apparatus must be rightly focussed in order to receive a sharply defined picture upon its sensitive plate, so the human eye must be correctly focussed in order to receive clear images on its retina. In the photographic camera the focussing is only a question of the proper adjustment of a single lens, but in the eye quite a number of refractive media have to be taken into account: the cornea, aqueous, lens, vitreous, and their surfaces. If we wish to know whether this dioptric system is correctly, i.e., normally, focussed we must know the path followed by the rays of light through it, and this we learn by physiological optics, as has been shown by *Donders* and *Helmholtz,* when we take into calculation accurately the curvature of the refracting surfaces, the distance from one of these to another, and the index of refraction of each of the refractive media, but this problem is too complicated for daily practical purposes, so we use the so called *reduced eye* of *Donders* to facilitate the calculation.

We understand by a reduced eye one which has an axial length of 20 mm and is composed of a single refractive substance having an index of refraction of 1.33. Its anterior surface, corresponding to the cornea, has a radius of curvature of 5 mm, therefore the centre of curvature is 5 mm behind the refracting surface and 15 mm in front of the retina. The centre of curvature is at the same time the nodal point of the eye. The *nodal point* is that point in the dioptric system through which the rays of light pass without being deflected. The anterior focal distance of this reduced eye is 15 mm, the posterior 20 mm, hence the retina coincides with the posterior focal distance.

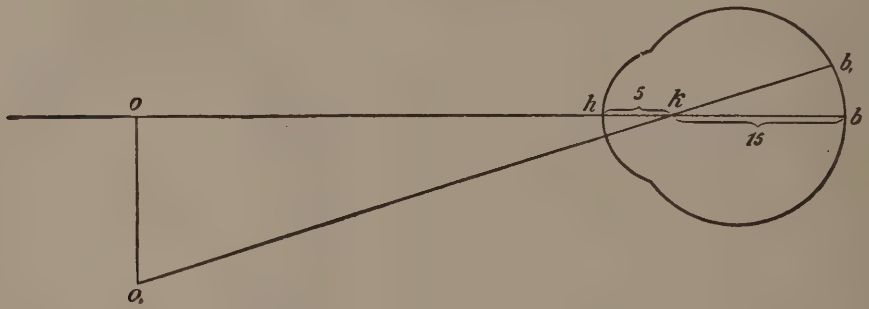


FIG. 143.—The Reduced Eye of *Donders.*

We may use the constants given in the following table to compare the actual conditions with those of this reduced eye.

CONSTANTS OF THE EYE ACCORDING TO *Helmholtz* (rather schematic)

Radius of curvature of the anterior surface of the cornea	8 mm
Radius of curvature of the anterior surface of the lens	10 mm
Radius of curvature of the posterior surface of the lens	6 mm
Depth of the anterior chamber	4 mm
Thickness of the lens	4 mm
Thickness of the vitreous	16 mm
Anterior focal distance	12 mm
Posterior focal distance	24 mm
Index of refraction of the cornea	1.33
Index of refraction of the aqueous	1.33
Index of refraction of the vitreous	1.33
Index of refraction of the lens	1.43—1.44.

The reduced eye differs from the normal in certain important particulars; the axial length of the normal human eye is 24 mm, that of the reduced eye 20 mm, yet we can confidently base our calculations on the reduced eye, for the results obtained by those calculations are extremely close to those based on the conditions of the actual eye.

Our first duty is to learn the course of the rays of light through the normal human eye. The problem is very simple in the reduced eye, which has only a single refracting surface and has the retina coincident with the principal focal point. Parallel rays of light are so refracted that they unite at a single point on the retina.

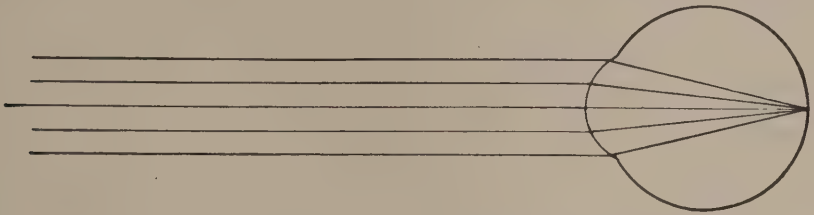


FIG. 144.—The Reduced Eye as an Emmetropic Eye.

When the posterior focal point for distance, i.e., for parallel rays of light that enter the eye, coincides with the percipient layer of the retina we call the eye normal, or *emmetropic.*

By the *refraction* of an eye we understand its optical condition when it is in a state of rest, i.e., when the accommodation is not acting. The refraction of a normal or emmetropic eye is therefore such that a sharply defined image is cast on the retina when the eye is adapted to infinite distance. In other words: *Emmetropia is present when the parallel rays of light that fall upon the cornea are united without assistance to form a sharply defined image on the retina.*

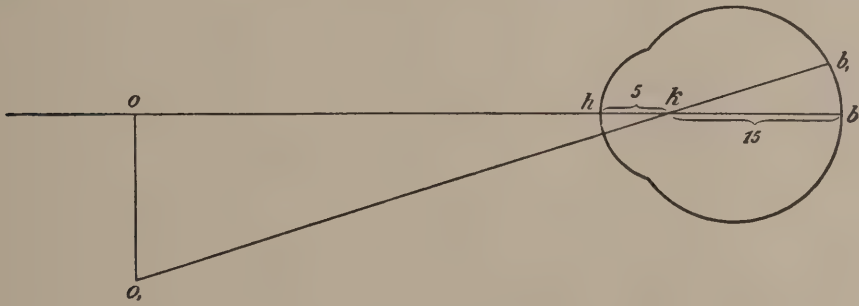


FIG. 145.—Calculation of the Size of the Retinal Image in the Reduced Eye.

We can solve another problem while following the rays of light in the reduced eye: we can calculate the *size of the retinal image.* Let us take an example.

The rod *o o′* in front of the eye gives rise to the retinal image *b b′*. We can find this retinal image very simply by drawing straight lines from the terminal points of the object through the nodal point of the eye to the retina. The triangles *o o′ k and b b′ k* are similar. Therefore the size of the retinal image is to the size of the object as the distance of the nodal point from the retina is to the distance of the object from the nodal point of the eye. The values in the reduced eye are known to us: the distance from the nodal point to the retina is 15 mm, the radius of the cornea is 5 mm. The size of the object and its distance from the nodal point can be measured. We can therefore easily calculate the size of the retinal image of an object with the help of the reduced eye. Take, for example, a rod 1 metre long placed just 15 metres in front of the nodal point of the eye and we find the size of the retinal image to be

$$x : 1000 \text{ mm} :: 15 \text{ mm} : 15000 \text{ mm}$$

$$x = \frac{15000 \text{ mm}}{15000 \text{ mm}} = 1 \text{ mm.}$$

This problem, to calculate the size of the retinal image, does not come within the province of the practicing physician. The most important problem for both him and the ophthalmologist to solve is whether the eye undergoing examination is normal or not. An eye may be said to be normal or emmetropic if parallel rays unite to form a sharply defined image on the retina when it is adjusted for distance and no other processes are taking place within it. It may be said: That is stated well enough mathematically or physically, but how are we to know whether such is the case with our patients?

We have various methods at our disposal by means of which to ascertain the optical construction or the refraction of an eye, some of which are dependent on the cooperation and the statements of the patient, while some are independent of them. The first thing to determine is the *visual acuity.*

A certain degree of light sense is requisite to enable us to recognize an object, i.e., the eye must be able to perceive the differences in the brightness of contiguous surfaces. The eye must also be able to perceive the form of a contour and to recognize the details of an object. This function is independent in many respects of the light sense. We may call it with *Hering* the optical sense of space, or the optical power of resolution of the eye.

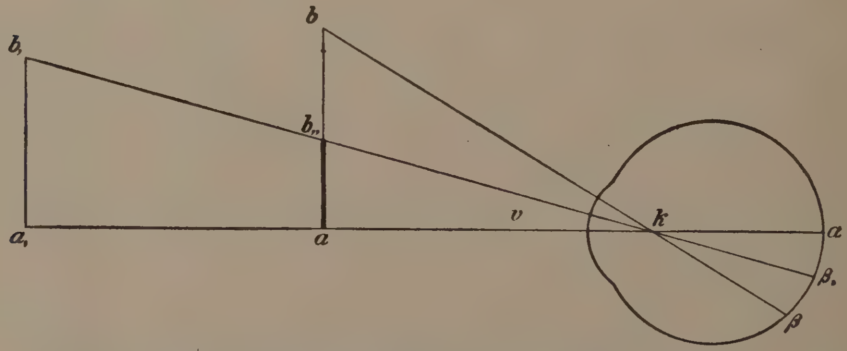


FIG. 146.—Method of Determining the Visual Acuity.

In the third place the eye must be able to interpret correctly the sensations excited in order to recognize an object; here we enter the realm of the intelligence of the mind. Of these three factors the optical sense of space is the most essential, and we may define visual acuity as the ability of the eye to distinguish separate points or lines. In order that we may learn to know, and above all to measure, this function of the eye let us study this simple drawing.

Suppose an eye is just able to see the rod *a b* which is placed 10 m in front of its nodal point; it has a certain visual acuity and the image of the rod $\alpha\ B$ is formed on its retina. We find the size of this retinal image by drawing straight lines from the terminal points of the object through the nodal point of the eye. These lines enclose an angle which we call the *visual angle,* or the *angle of vision.* Another man is able to see the same rod at a distance of 20 m; his eye evidently has the better vision. The retinal image in his eye is only one half the size of that in the other, $\alpha\ B'$, and the visual angle is also reduced one half. Instead of removing the object farther from the eye we may make it smaller. If *a b* is reduced in size one half, to $a\ b''$, the retinal image is correspondingly reduced in size. A third eye may be able to see the object when it is still smaller, or when it is removed still farther away—the vision of that eye is still better.

The acuity of vision is determined by the smallest angle with which an object can be seen. The smaller the angle the smaller the retinal image, the greater is the visual acuity. *The visual acuity is inversely proportional to the angle of vision.*

The interval between two points at a certain distance which just enables them to be perceived separately is called the *minimum separabile.*

The purpose of the test of vision is to determine the limits fixed for the sense of form, the ability to distinguish two points or lines. There is still one thing more that we must have before we turn to the practical estimation of the visual acuity, and that is a *unit angle.*

This unit angle is the *visual angle of 1 minute. Snellen* ascertained that the angle of 1 minute was the smallest visual angle with which two points could still be perceived to be separate by a number of normal eyes, and the practical determination of vision is based upon this fact. The vision of an eye is the sharper and better the farther away or the smaller are the objects that it is able to see. Hence we have two ways in which we may test the visual acuity: we may either take an object of a given size, remove it farther and farther from the eye and ascertain the distance at which it can just be recognized, or we may keep the distance constant and present smaller and smaller objects until we have ascertained which is the smallest that can be correctly perceived by the eye.

Letters and figures have long served as suitable test objects for the determination of the visual acuity. The test types based on the principle enunciated by *Snellen* that the visual angle of 1 minute is the smallest with which two points can be distinguished as separate, and known as *Snellen's* test types, are the most commonly used. Each letter is inscribed in little squares. Near the top letter we see the inscription D=60 m. This means that an eye with normal vision can recognize this letter at the distance of 60 m, the distance at which the entire letter subtends a visual angle of 5 minutes and each individual stroke of the letter an angle of 1 minute. We may, therefore, say that an eye which can distinguish the details of this letter at 60 m has normal visual acuity.

Suppose now that we take a patient who has a dense corneal opacity in one eye, with which he cannot recognize this letter at 60 m, but can when it has been brought as near as 6 m. The vision of that eye is less than normal; instead of 60/60 it is only 6/60. This gives us the formula for the expression of our findings by the test of the vision.

We express the visual acuity of an eye by a fraction, the numerator of which gives the distance at which a letter of the Snellen test types can actually be read, the denominator the distance at which it should be read by the normal eye. To express the same idea differently: The visual acuity of an eye is the distance at which two points can be distinguished as separate divided by the distance at which they subtend an angle of 1 minute. This gives the formula V=d/D, d denoting the number of metres at which the reading is done, D the number of metres at which the letter could be read with a normal eye.

Let us take another example: An eye that reads the letters of the second line, which should be recognizable normally at 30 m, only at the distance of 6 m has a vision not of 30/30, but of 6/30. It would be quite impracticable, not to say impossible, in our rooms to test the vision at such great distances as 60 and 30 m, so the first method of testing the vision, by moving an object to a greater distance, is used in practice only when the visual acuity of an eye is so low that the largest letter of the test types cannot be recognized at the distance of the length of the room. In such a case we hold our outstretched fingers in front of a dark background, ask the patient to count them and advance toward him step by step until we have ascertained the number of metres at which he is just able to see and count them. The width of the lines of the largest letter on the chart is approximately the same as that of each of our fingers. Here is a patient with a grave disease of the retina. He cannot see the largest letter of the test types at 6 m, so he has not 6/60 of visual acuity. I hold my fingers up before him and find that he can count them at 2 m, so we may say V=fingers at 2 m, or approximately 2/60. Sometimes we need to add fingers excentrically, to the temporal or to the nasal side, upward or downward.

As a general rule, we use the second method of testing. We have the patient read the letters on the test card, beginning above with the larger ones, keeping the distance unchanged. This patient reads all of the letters on *Snellen's* test chart, even the smallest. His vision is therefore 6/6, or normal.

The distance at which we make the test is of no particular consequence. We are working with a chart adapted to a distance of 6 metres, but as every physician has not as long a room as this at his disposal, test charts have been devised and adapted to 5 m, 4 m, and 3 m, but the test should not be made at a shorter distance than 5 m, because the accommodation should be relaxed while we are making the examination, and this relaxation is obtained only when the eye is looking at an object in the distance. When the accommodation is relaxed the glass necessary to give distinct vision in the distance is also the correction of the abnormal refraction of the eye.

It has already been stated that these test types are based on the principle that the visual angle of 1 minute is the smallest with which it is possible to perceive two points to be separate, or with which the form of a given object can be recognized. Although this method, which is in daily use, gives the impression that it is very exact, it is not, and this has been known for many years.

The first question that arises in this connection is whether the angle of 1 minute is actually the smallest on the average with which two points can be distinguished to be separate by the normal eye. We often meet with young persons whose visual acuity is much greater than can be expressed by 6/6. Cases in which the vision is one and a half or twice the normal are very common. *Cohn* has seen eyes in which the vision was six and eight times the normal. Hence it is possible for us to have a patient with a considerable impairment of vision whose visual acuity is 6/6, if previously he had 6/4 or 6/3. The frequent occurrence of low degrees of astigmatism especially has not been taken into account in the construction of *Snellen's* test types. Hence a vision of 6/6 does not represent the best vision of a normal eye, but only an average value which suffices for most of the demands of ordinary life.

If we should improve our test types by taking the maximal ability of a normal eye as the unit we would place ourselves in an unpleasant situation, for we would be obliged to call the vision of the overwhelming majority of people subnormal, as a visual acuity of eight times *Snellen's* unit is an extremely rare exception. The minimum of the visual power of normal eyes is likewise not useful as a unit, because the vision of most normal eyes is better than this. The only thing that is left is to establish a new average value by the examination of many emmetropic eyes, and this results in a unit that is higher than *Snellen's*, because the fact is that most emmetropic eyes are able to do more than *Snellen* thought.

A second disadvantage of the test types hitherto in use is this: *Snellen* started from the hypothesis that just as large a visual angle was needed for the recognition of the individual parts of a letter as for that of a point, but this hypothesis is not correct. Other factors enter into the recognition of letters besides the smallest visual angle. Their recognition does not depend wholly on the distances between the points and lines of which the test letters are composed, but relies a great deal on their forms and areas. Under certain circumstances the entire impression of the form of a letter is not obtained through the retinal image alone, but through the movements of the eye carried along its contour. These facts have not hitherto been taken sufficiently into account in the construction of most of the test types used, and this explains how it is that their most striking fault, the one most generally recognized, is that a row of characters of the same size are by no means uniform in the ease or difficulty with which they can be deciphered. In other words, characters of the same size do not make equal demands upon the power of vision. There are some small characters that can be recognized when other larger ones cannot. Hence objects of the same size and seen with the same visual angle are not discerned equally well. The discernibility of all of our letters is dependent on a considerable number of factors. It is evident that such difficulties render it impossible to ascertain accurately the true power of perception of an eye that is undergoing examination. This source of error in so essential a method of examination has long been recognized, and all manner of suggestions have been made to improve our test types, but a real improvement has not yet been obtained. This is shown by the fact that most widely different results, that cannot easily be compared together, are obtained with different test types in one and the same eye.

The best test types yet obtained are *Landolt's* black rings, interrupted at certain places by a space with parallel margins, printed on a white ground.

An international commission under the presidency of *Hess* has of late been studying the problem how to minimize the sources of error in our test types and to suggest a uniform test. My clinic has contributed a proposal to this international commission, based on special researches. In a scientific sense we have as yet no uniform, unobjectionable method of ascertaining the visual acuity. We still adhere to figures and letters, which have been found to furnish a more uniform measure of the power of perception than other characters, but it is necessary that the sources of error in the old test types should be eliminated. These sources of error are: 1, the average vision of the normal eye has been too much underestimated in the charts heretofore used, and 2, the characters used in the same line cannot be deciphered with sufficient uniformity. In addition to this the results obtained by an examination with one chart cannot be compared with those obtained with another. This can be shown best by means of the following table, which was compiled by my assistants, *Gebb* and *Loehlein*.

The characters nominally to be read at 5 metres in the test types of	Actual average distance of perception for emmetropic eyes								
	11 m	10 m	9 m	8 m	7 m	6 m	5 m	4 m	3 m
Heine	—	—	714	2	0 3	8	—	—	—
Schweigger	—	—	L	7 0	43P6 2T E	5 C	—	—	—
Roemer	—	—	—	—	—	—	65UD RS7 L	—	—

In view of these difficulties it seems to be certain that there is only one way in which to obtain a satisfactory chart. We must take as a measure of the normal visual acuity a size corresponding as closely as possible to the actual conditions, after it has been demonstrated that the *average* visual acuity of emmetropic eyes is greater than it has been supposed to be. We must place together in a row only such characters as have been proved to be perceptible to normal eyes with about the same degree of ease, when the choice of the characters by the smallest visual angle alone has not fulfilled this postulate. The test chart that I use combines these two principles.

The test types should be illuminated by constant artificial light, for *Cohn* has proved that the daylight may change in a few minutes from 19 to 106 metre candles.

The chart is made in a quadratic form, because in this way a uniform illumination is best guaranteed. Now that we know that the visual angle is not the only decisive factor in the recognition of letters, figures and forms we will substitute the term vision for visual acuity and the formula then reads V (Visus, acuity of discernment)$=d/D$. The chart is placed at the distance of 5 metres, and with it we test each eye of the patient in turn. If the patient can read only the top row, $V=5/50=0.1$ of the normal vision. This is the principle of one of the most important examinations of the eye that we have to perform, the testing of the vision, and this is the way in which it is done. Each individual test of the vision serves primarily to answer the question: Is this eye normal or not? It also tells us whether the visual function of the eye is normal or not, for if its visual acuity is perfect its functional power is normal.

190. The Three Conditions of Refraction of the Human Eye and Their Determination in Practice through the Vision

The human eye has a normal construction, or has an emmetropic refraction, if parallel rays are focussed on its retina when it is adjusted for distance and the accommodation is not called upon for assistance, that is, when the retina lies in the principal focal distance of the dioptric system. The point for which the eye is focussed when in a state of rest we call the *far point of the eye* (*P. R.*=punctum remotum). This far point lies at an infinite distance from an emmetropic eye.

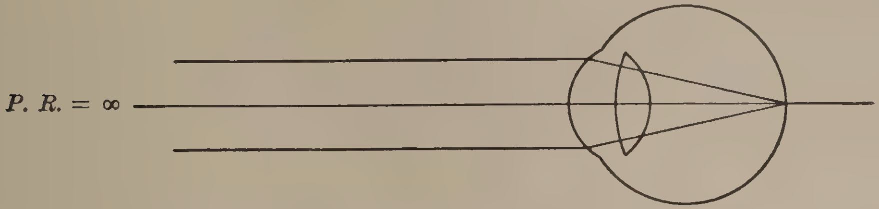


FIG. 147.—Course of the Rays of Light in Emmetropia.
P. R. = ∞

Two principal deviations are possible from the normal or emmetropic refraction: the eye may be too long in proportion to its refractive power, when we call it nearsighted or myopic, or it may be too short, when we call it farsighted, or hypermetropic.

Course of the Rays of Light in Myopia

When an emmetropic eye is in a state of rest parallel rays of light are focussed on its retina so as to form there a sharply defined image, and the eye sees distant objects clearly, but when the eye is myopic, that is, when it is too long in proportion to its refractive power, these rays are not focussed on the retina. Their course can be readily understood from a study of these drawings.

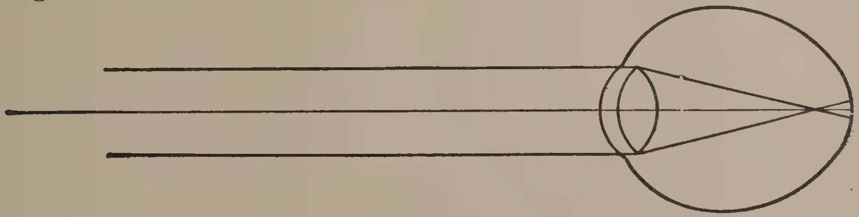


FIG. 148.

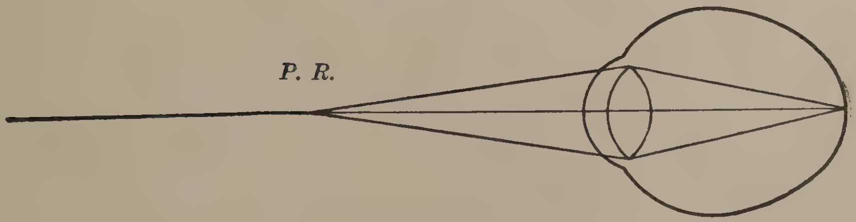


FIG. 149.

Course of the Rays of Light in Myopia.
Far Point (*P. R.*) at a Finite Distance from the Eye.

Parallel rays of light that enter the myopic eye while in a state of rest must intersect in the vitreous, cross, become divergent and fall upon the retina in diffusion circles; hence the eye cannot see distant objects clearly. The only rays that can be focussed on the retina are those that are divergent and come from a point situated at a finite distance from the eye, which is its far point. The vision of such an eye for all objects situated beyond this point is indistinct, it is distinct only for objects that lie only a short distance away, hence the name shortsightedness, or nearsightedness. The far point of the myopic eye is of interest for two reasons.

1. The nearer the far point lies to the eye the greater is the degree of myopia, for the farther in front of the retina the parallel rays intersect the more indistinct are the retinal images and the larger are the diffusion circles. The degree of myopia can therefore be determined through the position of the far point.

2. The determination of the far point gives us not only the degree but also the correction of the myopia, for as the rays that come from the far point, which is situated at a finite distance in front of the eye, are the only ones that can form a distinct image on the retina when the eye is at rest, we shall be able to make such an eye see distant objects clearly only when we give the parallel rays coming from those objects the same degree of divergence as those that come from the far point. Now we need to make use of what we have learned in regard to spectacle glasses. Suppose that we have a myopic eye and that we find the far point to be situated 25 cm in front of it. I will hold a concave glass of 4 D in front of it as it looks into the distance. The parallel rays of light that fall upon the glass will be rendered divergent, as though they came from its focal point; but this focal point lies 25 cm in front of the lens and therefore coincides with the far point of the eye. Hence the parallel rays of light are so directed as to be focussed into a sharply defined image on the retina of the nearsighted eye, which immediately sees distant objects clearly because its nearsightedness is corrected. From this we learn two facts: *The myopic eye sees distinctly at an infinite distance with the concave glass which has a focal distance equal to the distance of the far point from the eye.* We disregard for the present the distance of the glass from the vertex of the cornea. Reversing the above proposition we may say: *The focal distance of the concave glass with which the myopic eye is able to see distinctly at a distance gives us the position of the far point and the degree of myopia.* In the example given a concave glass of 25 cm focal distance is needed and the eye has a myopia of 4 D.

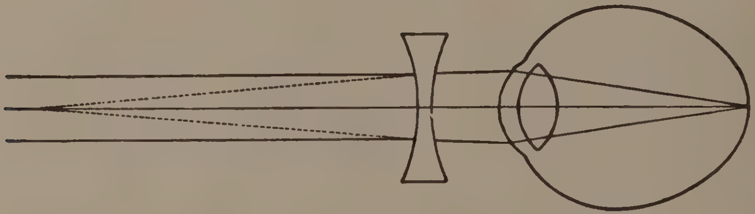


FIG. 150.—Correction of Myopia by a Concave Glass. The Parallel Rays That Fall upon the Glass Are Given the Same Direction as though They Came from the Far Point of the Eye.

The second deviation from the normal, emmetropic refraction is hypermetropia. The chief cause of myopia is an optic axis that is too long, that of hypermetropia is one that is too short.

The course of the parallel rays of light which fall upon the cornea is shown by the accompanying diagram, which shows that parallel rays are refracted so that they would intersect behind the retina. For the sake of simplicity we will assume that the cone of rays of light is not cut off by the retina before it reaches its apex. If we repeat the experiment with divergent rays, drawing an object from infinite distance toward the eye, we find that the rays focus farther and farther behind the eye and that the circles of diffusion constantly grow larger. The hypermetropic eye is so constructed that when it is in a state of rest such rays only are focussed on the retina as fall upon it with a certain degree of convergence. The refractive power of the dioptric system is able to focus upon the percipient layer of the retina such convergent rays alone. If these convergent rays that enter the eye are prolonged we find that they will intersect at a certain point behind the eye, and we call this the far point. Hence the far point of a hypermetropic eye is behind the eye. There is no true point of intersection of these rays, we can only note their direction, and we accordingly indicate the far point with the negative sign: – R. Now the question is: How shall we be able to focus parallel rays of light on the retina of a hypermetropic eye? It is evident that such rays must be more strongly refracted, must be gathered together, that the refracting power of the eye must be aided.

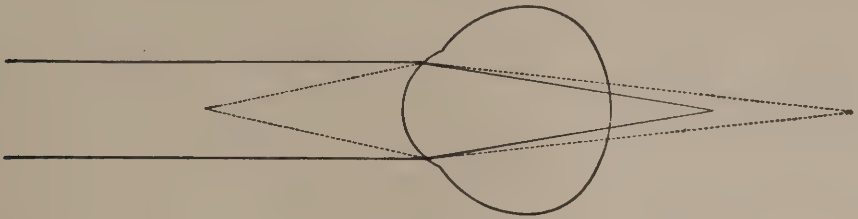


FIG. 151.

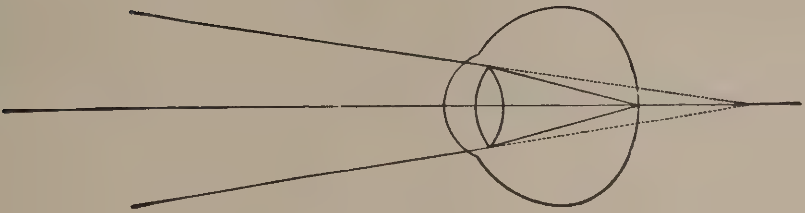


FIG. 152.
Course of the Rays of Light in Hypermetropia.

In youth the hypermetropic eye can help itself by increasing the curvature of the lens by means of the mechanism of the accommodation. The zonule is relaxed in consequence of a contraction of the ciliary muscle and then the lens is compelled by its elasticity to assume a more nearly spherical form and its surface becomes more curved. When the hypermetrope wishes to see distinctly at a distance, to focus parallel rays of light upon his retina, he must give his lens an increased degree of curvature. We may do this work for him and substitute for this increased curvature of the lens a suitable convex glass held in front of the eye, which represents to a certain degree the same additional lens power that the eye otherwise produces spontaneously by means of the accommodation. The strength of this convex lens is dependent on the distance of the far point. Let us suppose that the far point of a hypermetropic eye is situated 25 cm behind it. The only rays that can be focussed a priori on the retina are those that enter the eye with such a degree of convergence that they will meet at that point when they are prolonged. What convex glass do we need to enable parallel rays to be focussed in this manner? Evidently a glass of +4 D, as it has a focal distance of 25 cm. If we neglect again the distance of the glass from the cornea such a lens will give the rays of light the same direction as if they came from the far point of the hypermetropic eye. The focal point of the lens and the far point of the eye coincide. Hence in hypermetropia *the focal distance of the glass with which the hypermetrope sees clearly at a distance, or with which parallel rays of light are focussed on his retina, gives us the degree of the hypermetropia.* Everywhere and always we meet with the importance of the far point, which may be thus defined: *The knowledge of the position of the far point of an eye gives us at once its optical construction in a state of rest*, in other words, *its refraction.* We know that if the far point of an eye lies in infinity, that is if parallel rays of light are focussed on the retina without any assistance from within or without the eye, we can say at once that this eye is emmetropic, or normally constructed. If the far point is situated at a finite distance in front of the eye we know the eye to be myopic, and if the far point is behind the eye we know the case to be one of hypermetropia. This is sufficient for the theory, we will turn now to its practical application.

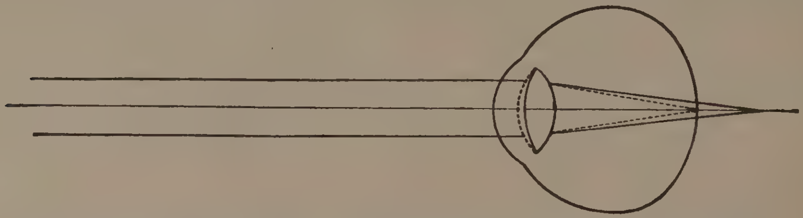


FIG. 153.—Self Correction of Hypermetropia by the Accommodation.

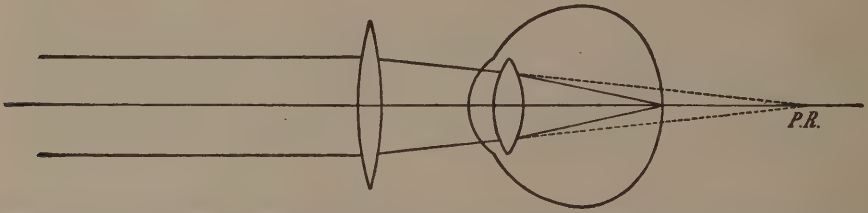


FIG. 154.—Correction of Hypermetropia by the Interposition of Convex Glasses.

The first step toward learning what is the refraction of an eye, i.e., toward determining its far point, is to test the vision. We place the patient at the proper distance in front of the test types and then the first question to be answered is whether he is emmetropic, hypermetropic, or myopic.

The patient reads all the characters on the chart, including the row D=5, therefore V=5/5=1, he has perfect visual acuity. I must call attention to one point: *the rays from the small letters on the chart at this distance form so small an angle when they pass through the pupil of the eye that we consider them practically parallel, or as coming from an infinite distance.* Thus we are dealing here with the parallel rays of which so much has been said.

First of all, we know that this eye cannot be myopic, for parallel rays cannot be focussed on the retina of a myopic eye without proper correction, but must create circles of diffusion. Hence the eye must be either emmetropic or hypermetropic.

A hypermetropic eye can focus parallel rays of light on its retina, that is, it can see well at a distance, as long as it has at its disposal a sufficient amount of accommodative power and is able to increase the imperfect refractive power of its dioptric system by causing the lens to assume a more spherical form. If the patient is hypermetropic he can see the lowest row of letters, D=5m, i.e., he can focus parallel rays upon his retina, only when he increases the curvature of his lens somewhat by means of his accommodation. The hypermetrope does this only when it is necessary in order to obtain distinct vision. We can save him a part of the effort when we place a convex lens before his eye, as he then relaxes his accommodation, or calls upon it only for sufficient effort to enable him to perceive the characters distinctly with the aid of the convex lens.

The emmetrope cannot do this. If we place a convex glass before an emmetropic eye adjusted for distance the eye immediately becomes nearsighted and does not see well far away, because it can no longer focus parallel rays of light upon its retina. Hence, *when an eye has equally as good or better vision with a convex glass as without it, it is hypermetropic.*

When I hold a +1 D glass in front of the eye of this patient he says at once that it impairs his vision, that he can no longer read line D=5. This proves that his eye is neither myopic nor hypermetropic, but emmetropic with a vision of 1. His far point is at infinite distance in front of his eye. The test of the vision always amounts to the determination of the far point and of the nature of the refraction of the eye.

191. The Mechanism of Accommodation in the Human Eye and the Determination of the Amplitude of Accommodation

This boy, ten years old, has normal, emmetropic eyes, and the visual acuity of each is likewise normal. Atropin has been instilled in one eye, the pupil of which is dilated and no longer reacts to light. But it is not the pupil alone that has been paralyzed. If the boy is asked to read first with one eye and then with the other we find that he is unable to do so with the eye that is under the influence of atropin. The important mechanism of which we make use every minute and by means of which we are enabled to see near objects has been done away with. This mechanism is that of the accommodation.

By accommodation we understand the ability of the eye to focus upon objects at different distances in the interest of distinct vision, so that a sharply defined image will be formed on the retina. Every physician should be well acquainted with this process, not only because of its practical importance, but also on account of its scientific interest.

We have three problems to solve in connection with this subject. First, we must know what actually takes place in the eye during accommodation. Second, we must get an idea how these changes come to take place. Third, we must learn how to determine and measure the power of accommodation in practice.

I. The Objective Changes in the Eye during Accommodation

When I ask the boy to look off at a distance with the eye into which atropin has not been instilled and then to look quickly at this pencil the pupil is seen to contract as the eye becomes adjusted for near. The contraction of the pupil is the sign of accommodation that has been known for the longest time. It is due altogether to the innervation of the oculomotor nerve and has nothing to do with the quantity of blood in the iris or ciliary body.

This contraction of the pupil is accompanied by an advance of the anterior vertex of the lens. This phenomenon may perhaps be seen when we look at the eye in profile, but it is to be observed more distinctly with the binocular loupe. In order to give an idea of how much the anterior vertex of the lens advances, I may say that according to *Helmholtz* the advance amounts to 0.4 mm in persons who are young and have a strong power of accommodation.

The changes in the peripheral portions of the anterior chamber are less marked, yet the peripheral parts of the iris may sometimes be seen to recede a little.

The fourth symptom of accommodation is much more important. The anterior surface of the lens bulges strongly into the pupil, as can be seen with *Zeiss'* loupe from the anterior lenticular image, which appears as a clear, faded, shagreened looking spot, the granular aspect of which is due to the fact that we can see the capsular epithelium of the lens. The change of curvature of the posterior surface of the lens is much less important than that of the anterior. The lens itself increases in thickness during accommodation about 0.4 mm, according to *Helmholtz* from 3.6 to 4 mm. The changes of form in the peripheral portions of the human lens play no part in accommodation; it is only in the eyes of birds, as has been shown by *Hess* and *Fischer*, that the peripheral portions of the anterior surface of the lens are flattened by a lateral pressure of the ciliary processes.

Hess has also discovered that the lens shakes distinctly during strong accommodation, when little twitching movements are made with the eye. This shaking may be so great as to cause the iris to tremble. If the observer will watch the posterior lenticular image while the person employed for the experiment accommodates, it will be seen to make slight movements whenever the eye itself twitches. According to *Hess* the lens may also change its position during accommodation, during strong accommodation it sinks from the force of gravity from 0.25 to 0.3 mm. This sinking of the lens may be recognized entoptically. The spectrum of the lens may be seen entoptically by looking through a pinhole made in a card and is always found to contain little irregularities. By alternately covering and uncovering the little opening the contraction of the pupil can be perceived entoptically. If now attention is concentrated upon one of the entoptically perceived points in the lens it will be seen that after the contraction of the pupil the point in the lens rises during accommodation and falls again when the accommodation is relaxed. This apparent movement of the point in the lens upward corresponds, as is easily proved, to a sinking of the lens during accommodation.

All of these symptoms may be demonstrated still more distinctly when we instil eserin and so excite a strong contraction of the ciliary muscle, the muscle of accommodation.

It has been shown in eyes in which a defect in the iris had been produced by traumatism or operation, that the ciliary processes make a distinct movement forward toward the lens during accommodation. They are moved by the contraction of the ciliary muscle toward but not up to the plane of the equator of the lens and not backward. Occasionally in such eyes the fibres of the zonula may be visible as fine lines. The modern researches of *Hess* have also shown that the margin of the lens is slightly wavy in the atropinized eye, while in the eserinized eye it has rather a smooth circular form.

The measurements made by *Hess* on eyes that were treated with atropin and eserin shortly before death prove that the equatorial diameter of the lens during accommodation in a person 20 years old is from 0.4 to 0.5 mm less than its equatorial diameter when the accommodation is relaxed.

II. The Theory of Accommodation

The following theory of the mechanism of the accommodation was advanced by *Helmholtz*. "The crystalline lens is an elastic body upon which traction is made in a radiating direction by the tension of the zonula which is attached to its margin and is therefore somewhat contracted in the direction of its symmetric axis. . . . The radiating fibres of the ciliary muscle running in the direction of the meridian of the eye, which end in the tissue of the chorioid at the posterior extremity of the ciliary processes, draw forward the posterior end of the zonula, which is firmly attached to the chorioid and vitreous lamella at this place, and relax the tension exerted by the zonule upon the periphery of the lens, so that in consequence the lens contracts in the direction of its diameter and becomes thickened in the direction of its axis." *Hess* rendered a great service to ophthalmology by the researches in which he confirmed the correctness of the first part of *Helmholtz's* theory, by definitely proving that the increased curvature of the lens is due to a diminished tension of the zonule.

The ciliary muscle, or the muscle of accommodation, consists of two portions, the layer of circular fibres or *Mueller's* portion, and the layer of longitudinal fibres, or *Bruecke's* portion. The firm anterior insertion of this muscle lies at the corneoscleral margin, while its posterior end is lost in the chorioid. It is supplied by the oculomotor nerve. When the muscle contracts the superficial portion of the ciliary body and the most anterior portion of the chorioid are drawn forward. The circular muscle fibres are the principal ones called into action in accommodation and we consequently find this part of the muscle particularly well developed in eyes which have to accommodate a great deal.

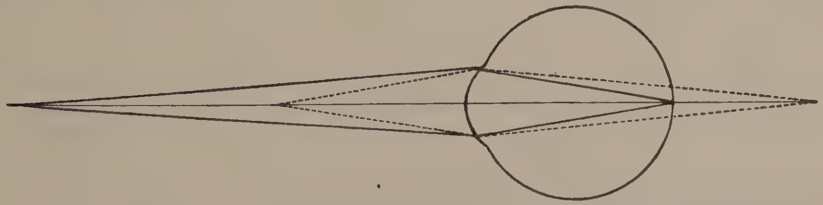


FIG. 155.—Circles of Diffusion when the Eye Is Incorrectly Focussed.

Both parts of the muscle cause by their contraction a forward movement of the ciliary processes and so a relaxation of the fibres of the zonule. As *Hess* says, the entire muscle acts as a "protractor corporis ciliaris." *Voelckers* and *Hensen* have shown this by demonstrating the forward movement of the chorioid when the muscle was stimulated by observing the movement of needles that had been stuck into the eye.

It is possible for one to convince himself of the activity of the ciliary muscle in his own eye; he can feel its action when he makes the following experiment. Close one eye, look at a book with the other and hold the point of a pencil between the book and the eye. The lines of print and the point of the pencil cannot be seen distinctly at the same time; either the print can be read and the point of the pencil seems blurred, or the point of the pencil can be seen plainly and the print is indistinct. If the pencil is held close in front of the eye and the focus is changed from the book to the pencil it will be felt that it takes an effort to do this, that a change takes place in the eye. This effort that is felt is the action of the accommodation. It may also be felt that the strain relaxes as soon as the fixation is changed from the pencil to the book. Anyone may convince himself by this experiment that a change of focus in the eye is produced by muscular activity—by the contraction of the ciliary muscles.

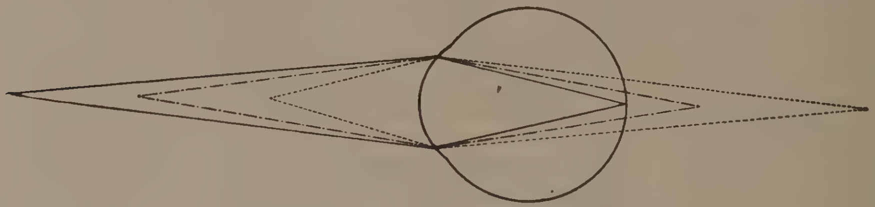


FIG. 156.—Vision in Circles of Diffusion.

The reason why the point of the pencil cannot be seen distinctly while the eyes are fixed on the book is very simple: the former is seen only in circles of diffusion. It may be readily seen from the diagram that rays coming from the point of the pencil have a greater divergence than those coming from the book, and must therefore intersect behind the retina as long as the adjustment of the eye is unchanged. Naturally the cone of rays is truncated by the retina before it comes to a point, and a circle of diffusion is therefore produced on it instead of a sharply defined image, because the base of the cone is formed by the round pupil.

We must always see in circles of diffusion when the eye is not correctly focussed upon the object of fixation. The larger these circles of diffusion are the more indistinct must be the vision, and their size depends mainly on the following factors.

If we interpose a third object between the point of the pencil and the eye the rays from this would intersect still farther back behind the retina and the cone of rays will be truncated by the retina still farther from its apex. *The circle of diffusion is therefore proportionally greater as the adjustment of the eye is the more inaccurate.*

Further, it can be easily shown that the size of the circles of diffusion is dependent on the size of the pupil. The transverse section of the cone of rays is smaller when the diameter of the pupil is less, as may be seen from this drawing. I mention these phenomena because they are of practical importance. The fact that myopes seem to see better at a distance as they grow old is partly due to the contraction of the pupils in advanced age. Elderly far sighted persons often desire a bright light because the circles of diffusion have been made smaller by a contraction of the pupil.

The primary cause of the change that is constantly taking place in the optical activity of the eye is the work of the ciliary muscle by means of which a relaxation of the zonule is brought about. The question naturally arises: How does the relaxation of the zonule affect the lens? A young lens is very elastic and its elasticity depends mainly on the constitution of the lenticular capsule. Under normal conditions, with the accommodation excluded, the fibres of the zonule exert a uniform traction upon the capsule by which the lens is kept flattened, but as soon as this tension of the zonule is relaxed the young lens, which is able to accommodate, immediately tries to assume a spherical form. We see this in patients whose zonule is torn in consequence of luxation or subluxation; the lens becomes more globular and causes an increase of the refraction, a myopia.

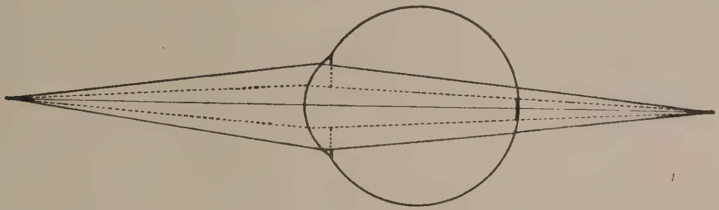


FIG. 157.—Dependence of the Size of the Circles of Diffusion upon the Diameter of the Pupil.

This tendency of the lens to assume a spherical shape after the zonule has been ruptured is to be seen still more clearly when we have an opportunity to examine this organ after it has been extruded from the eye of a young child, through the perforation made by a gonorrhœal ulcer of the cornea. Such a lens is almost in the form of a ball, while we know that it is flattened in situ. The relaxation of the zonule in accommodation is brought about by the contraction of the ciliary muscle. As this muscle contracts the circle formed by the ciliary processes becomes smaller, the apices of the ciliary processes move forward, and then the lens is enabled to assume a shape in conformity with its elasticity, because the zonule has become relaxed by this movement; the lens takes on an increased curvature, the middle of the anterior chamber becomes shallower, the lens trembles slightly and sinks a little from its own weight. This is the way in which accommodation is effected in the human eye. The attempt has been made many times to explain the accommodative increase of curvature of the lens, not by the relaxation of the zonule, but, on the contrary, by an increase of its tension, as by *Mannhardt*, *Schoen*, and *Tscherning*, but all of these hypotheses have been disproved by *Hess.*

III. Determination of the Amplitude of Accommodation

The entire mechanism of accommodation consists of an increase of the refractive power of the dioptric system of the eye produced by an increased curvature of the lens. Our patient looks away off into the distance, and the next instant he has increased the refractive power of his eye so much by means of his accommodation that he is able to focus upon his retina the rays coming from an object situated near by.

We call that point in space from which a sharply defined image can be produced on the retina when the curvature of the lens is the greatest to which it can attain, the near point—punctum proximum, or *P*.

It must not be imagined that a proximal contraction of the ciliary muscle is necessary in order to focus the eye upon the near point; the lens attains its greatest curvature before the maximal contraction of the muscle has been reached.

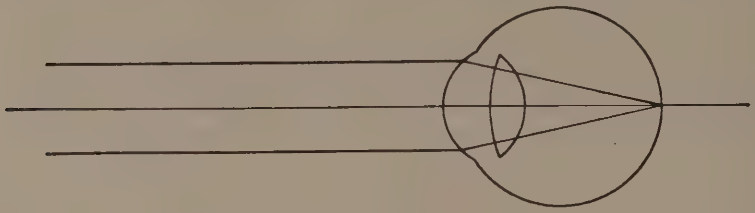


FIG. 158.—Looking at a Distance, the Accommodation Being Inactive.

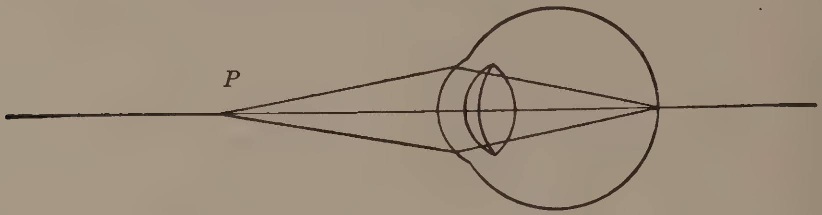


FIG. 159.—The Eye Focussed on the Near Point by the Accommodation.

The far point of the eye is that point upon which the eye is focussed when the curvature of the lens is least; it is also known as punctum remotum, or *R*.

From these two definitions it follows that we will find an expression for the accommodation employed if we are able to measure the increase of the refractive power which the eye undergoes in passing from the condition in which the accommodation is at rest (*R*) to that in which its tension is greatest (*P*). We call this increase of the refractive power of the eye the *amplitude*, or *range, of accommodation.* This amplitude we are able to measure.

As the curvature of the lens is increased in accommodation we may conceive of this as being accomplished by the interposition of a second small lens at the moment of accommodation, by which the refractive power of the dioptric system is increased. As may be seen from the accompanying diagram this supplemental lens represents the increase in the refractive power and furnishes the desired measure of the accommodation exerted. We cannot determine directly the refractive power of this supplemental lens imagined to be present in the eye, but we have convex lenses in our test cases, and we can hold them before the eye of this boy in which the mechanism of accommodation has been paralyzed by atropin. He reads at 10 cm with the eye that has not been atropinized, but cannot read at all with the other. If I hold a convex lens of 10 D in front of his atropinized eye he can read with it at once at the distance of 10 cm. Hence it follows that he was able to increase the refractive power of his normal eye 10 dioptres by means of his accommodation, and therefore that his amplitude of accommodation is 10 D. To state this in the form of a general rule: *We can express the amplitude of the accommodation by the convex lens which is able to give rays coming from the near point the same direction that they would have if they came from the far point.*

In this emmetropic boy the far point lies in infinity and the rays coming from it are parallel when they strike the eye. If we hold a +10 D lens in

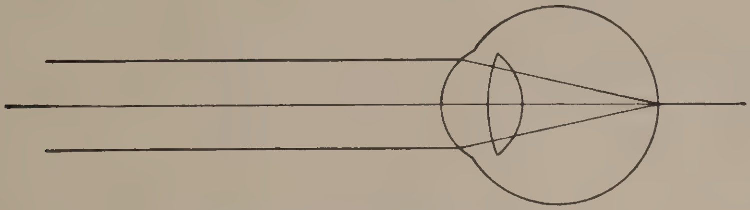


FIG. 160.—The Accommodation at Rest.

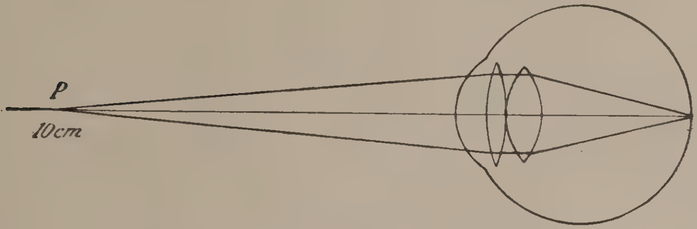


FIG. 161.—Imaginary Supplemental Lens to Represent the Accommodation.

front of the eye that is under the influence of atropin the divergent rays coming from his near point assume such a direction after they have passed through the lens that they are parallel when they strike the eye, as may be seen from this diagram. We neglect the distance between the lens and the eye, for this is of no importance in our practical work.

In order to be able to apply the above general formula for the determination of the amplitude of accommodation we need only to know how to ascertain the near point of any particular eye. There are scientific and practical methods of doing this. In our practical work we generally determine the near point by bringing fine print closer and closer to the eye until it begins to appear indistinct. The accommodation acts up to this point. The distance of the near point from the eye varies with the age and with the refraction. When we have reached the point at which fine print becomes indistinct we measure its distance from the eye and so find the position of the near point. This gives us the general formula for the determination of the amplitude of

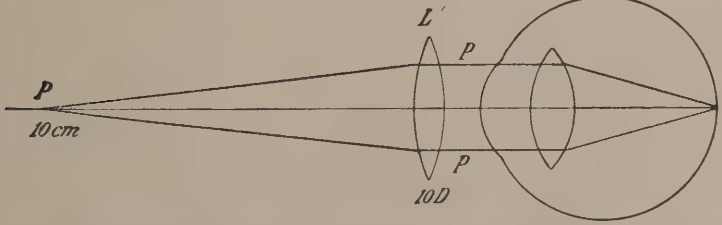


FIG. 162.—Substitution of a Convex Lens for the Imaginary Supplemental Lens.

accommodation. *Since the amplitude of accommodation represents the increase of the refractive power of the eye in the transition from the condition in which the accommodation is at rest to that in which its tension is greatest, we are able to measure it when we calculate in dioptres the difference between the refractive powers of the eye in these two conditions.* This is the general formula $A=P-R$, in which we have to express the values of the far point and near point in dioptres. The eye of this boy is emmetropic, he has no accommodation for distance, hence the value of R is 0; P is situated in his case at 10 cm. This corresponds to a convex glass of $100/10$ cm$=10$ cm focal distance, i.e., to a lens of 10 dioptres. Hence when the refraction is emmetropic $A=P$, in this case 10 dioptres.

The amplitude of accommodation can be calculated in the same way in any case of hypermetropia or myopia from the formula $A=P-R$. This patient has 2 D of hypermetropia. His far point is therefore situated 50 cm behind the eye and we indicate it with a negative sign. Such a hypermetrope must accommodate 2 D for distance in order to see clearly. Fine print begins to become indistinct 20 cm away. P therefore is situated 20 cm in front of his eyes, which corresponds to a convex lens of $100/20$ cm focal distance $=5$ D. His amplitude of accommodation is therefore $A=P-R=5-(-2)=7$ D. Our third patient has a myopia of 10 D. The far point of this eye is at 10 cm in front, so we designate it as positive. By having the patient read we find that the finest print can be read up to 5 cm from the eye, so the near point is at 5 cm, which corresponds to a convex glass of $100/5=20$ dioptres. But the amplitude of accommodation is not 20 D, for the far point is not situated in infinity as in emmetropia. On the contrary, the behavior of this eye when the accommodation is at rest is like that of an emmetropic eye without the power of accommodation before which has been placed a lens of $+10$ D. We must therefore take 10 D as the value of R and subtract it from the value of P. Thus the formula $A=P-R$ serves also for the determination of the amplitude of accommodation in myopia. This myope has a range of accommodation of 10 D.

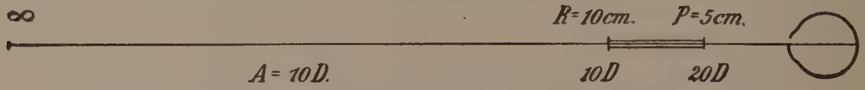


FIG. 163.—Region of Accommodation in a Young Myope of 10 D.

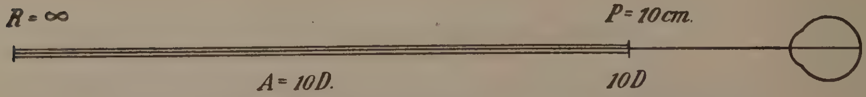


FIG. 164.—Region of Accommodation in a Young Emmetrope.

Let us compare our emmetropic boy with this myope. Both have an amplitude of accommodation of 10 D, but the distance between the far point and the near point is immensely different, as these diagrams show.

This distance in space between the far point and the near point we call the *region of the accommodation.* These linear extents include all points in space from which a distinct image may be formed on the retina with the aid of the accommodation. This space is very great in the young emmetrope, for it extends from infinity to a point 10 cm in front of his eye, while in the myope it is very small and has an extent of only 5 cm. The emmetrope can use his eyes at all distances at which distinct vision is needed in practical life, while the myope sees clearly only for a distance of 5 cm, which in addition is situated close to his eye. The emmetrope has the advantage as regards the utilization of the accommodation, but the region of the accommodation gives us no measure of the work done by the ciliary muscle, for the range of the accommodation is the same in both cases.

The range, or amplitude, of accommodation is due wholly to the physical behavior of the lens, which depends essentially on the age rather than on the construction of the eye, so that the range decreases regularly as age increases.

192. The Physiological Decrease of the Amplitude of Accommodation and the Prescription of Presbyopic Glasses

When this patient, who is advanced in years, is asked to read he holds the book far away from his eyes. He can no longer accommodate as he could when he was younger, so as to read with the print from 15 to 20 cm away. It has already been stated that the near point recedes as age advances, so that the range of accommodation constantly grows less. This decrease of the amplitude of accommodation is a purely physiological process, and we need first to inquire into its cause.

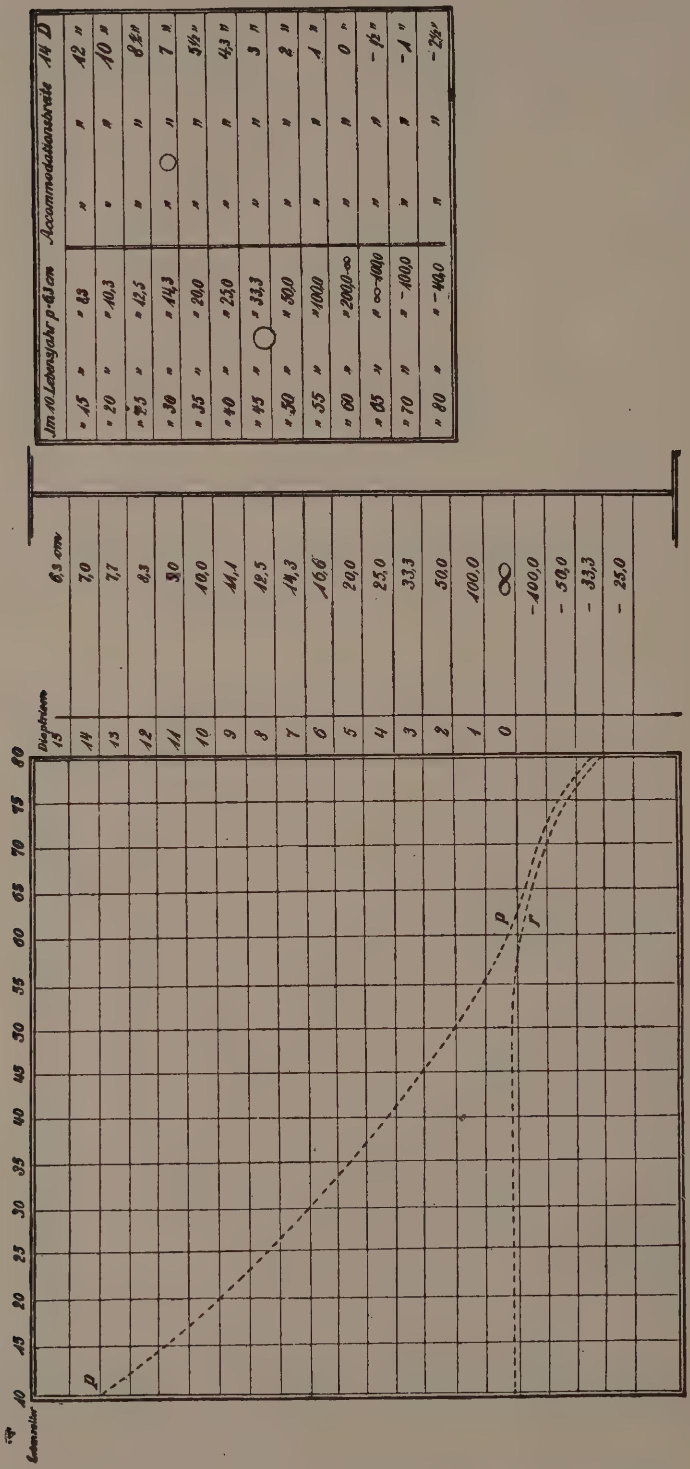
I. Cause of Decrease of the Amplitude of Accommodation as Age Advances

It must not be thought that the working capacity of the ciliary muscle decreases with the advance of age, for the decrease of the range of accommodation begins quite regularly in early childhood. The recession of the near point depends altogether on the fact that as age increases our lenses lose more and more their softness and elasticity. A nucleus forms in the centre of the lens at a comparatively early period. We find it distinctly developed in persons in the twenties. The central fibres of the lens become thickened in consequence of the process of sclerosis that is going on within it, the nucleus gradually grows larger, it is harder than the cortex and then the soft cortex alone is capable of a change in form when the zonule is relaxed in accommodation. Finally, in old age, the entire lens may be composed of a single firm nucleus, and then all power of accommodation has been completely lost.

A lens freshly taken from the eye of a child has a nearly spherical form and is very elastic, so when the ciliary muscle contracts in a young eye and causes the zonule to relax it immediately increases its curvature. But lenses taken from the eyes of elderly people are flattened and hard. No matter how strongly the ciliary muscle may contract in such an eye the lens can no longer arch its surface, because no soft cortex is present.

II. The Rule Governing the Diminution of the Amplitude of Accommodation

This recession of the near point, this loss of elasticity by the lens, this diminution in the range of accommodation, takes place with a remarkable uniformity from youth to old age so long as the eye remains normal. *Donders* has delineated this change in the accommodation at different ages in emmetropic eyes by the following curve:



Jm 10. Lebensjahr		p = 6,3 cm	Accommodationsbreite		14 D
" 15	"	" 8,3	"	"	12 "
" 20	"	" 10,3	"	"	10 "
" 25	"	" 12,5	"	"	8½ "
" 30	"	" 14,3	"	"	7 "
" 35	"	" 20,0	"	"	5½ "
" 40	"	" 25,0	"	"	4,3 "
" 45	"	" 33,3	"	"	3 "
" 50	"	" 50,0	"	"	2 "
" 55	"	" 100,0	"	"	1 "
" 60	"	" 200,0-∞	"	"	0 "
" 65	"	" ∞-100,0	"	"	-½ "
" 70	"	" -100,0	"	"	-1 "
" 80	"	" -40,0	"	"	-2½ "

Fig. 165.—Amplitude of Accommodation of an Emmetrope.

The line *r r* gives the position of the far point, the line *p p* that of the near point in emmetropia. On the ordinates are indicated the ages, on the abscissæ the distances in centimetres between the near point and the far point to the right of the corresponding values in dioptres. The distance between the two lines thus gives in dioptres the range of accommodation present at the different ages. For example: The far point of an emmetrope 25 years old is infinity, the near point is 12.5 cm from the eye, and the range of his accommodation is 8 D. When he is 50 years old his range of accommodation is only 2 D.

The far point also recedes somewhat from the eye in old age and the emmetrope becomes hypermetropic. It was formerly believed that the lens became more homogeneous as age advanced, that the index of the cortex increased more than that of the nucleus, but we know today that the difference between the indices of the cortex and of the nucleus does not decrease, but, on the contrary, increases considerably as the person grows older. Upon what the recession of the far point depends has not yet been sufficiently investigated. The actual conditions are of more importance to us. The regular course in the diminution of the amplitude of accommodation enables us at times to determine the age of the person being examined. When the age is too much understated the ophthalmologist needs only to determine the amplitude of the accommodation, as may be shown by an example. The near point of this emmetropic patient is situated at the distance of 20 cm, therefore he is about 37 years old.

III. Presbyopia

When in the course of life this diminution in the range of accommodation has reached such a degree that a man can no longer conveniently do his most important work, such as reading, writing, and fine handiwork, at the usual working distance a condition has developed which we call presbyopia. Reading is difficult, because the print when held far from the eye appears to be very small, though it is still possible to read it for a while when the light is strong, because the circles of diffusion become smaller with the contraction of the pupil. Reading is soon given up entirely at times when the light is not so good. Persons with presbyopia have no pain or other symptoms of asthenopia, such as we meet with in cases of hypermetropia, or of imbalance of the muscles of the eye, for the diminution of the accommodation in age is a physiological phenomenon. It seems to be impossible to make a certain age limit responsible for the beginning of presbyopia, for the onset of the trouble varies according to the vocations of different men and also according to the construction of the eye. Anyone who has been accustomed to do his work at a distance of 25 cm will become presbyopic sooner than one whose work is at a distance of from 33 to 45 cm. Apart from the vocation the development of presbyopia is particularly dependent on the construction of the eye itself. The hypermetrope who is continually obliged to use a part of his accommodation to enable him to see clearly at a distance must become presbyopic earlier than the emmetrope, and the latter sooner than the myope. Many myopes do not become presbyopic at all. Those persons who have a myopia of 3 or 4 D, whose far point is therefore at 33 or 25 cm, can still read at those distances without glasses even in old age, because they use for this purpose not the near, but the far point. Under such conditions the eye is exactly adapted to the working distance. Persons with lower degrees of myopia become presbyopic later than those with normal eyes. The knowledge of the range of accommodation is preconditional to our power to aid presbyopes by the prescription of proper convex glasses, with which we replace what they have lost. Emmetropes begin to complain of presbyopia, as a rule, between the ages of 45 and 50.

IV. Prescription of Glasses for Presbyopia

Certain data may be obtained from *Donders'* curve, which will be of help to us in the examination of patients with presbyopia and in the prescription of the proper glasses, but we cannot proceed schematically. Take a simple example: A carpenter, 60 years of age, has his work at a distance of 45 to 50 cm; we must not prescribe simply a glass of +4 D, because he has no accommodation according to the curve, but must prescribe a weaker lens for him to wear while he is at work, and, if need be, a stronger one for him to read with. Some rules may be given in regard to the prescription of such glasses, but a correct conception of how to do it can be obtained only by practice. First I wish to call attention to two erroneous views that are widespread among the laity concerning the wearing of presbyopic glasses. One is that the use of such glasses should be delayed as long as possible. Persons who believe this will not "pamper" their eyes for fear that they will the more quickly need stronger glasses. Such people struggle along for years without glasses to their own injury, for they cannot check the progress of their presbyopia. The process of sclerosis takes its course in the lens just the same whether they wear glasses or not. We cannot arrest it by exercise and straining of the ciliary muscles, for presbyopia is due wholly to the condensation of the lens. The ciliary muscles as such have nothing to do with it. Other people begin to wear glasses as early as possible in the hope that they may thereby conserve their eyes, but this expectation is unfounded for the same reason. *The first and main question in the prescription of a presbyopic lens is: Shall we give exactly the glass by which the near point is brought to the necessary working distance, or shall we prescribe a stronger one?* There are those who believe that a presbyope must exert a maximal contraction of his ciliary muscle when reading at his near point, so, as weariness of the eye cannot be prevented under such conditions, a stronger glass should be prescribed. But we know from the researches of *Hess* that the strongest contraction of the ciliary muscle is not needed to produce the greatest possible increase in the curvature of the lens. It is therefore not necessary to prescribe stronger glasses than are needed to enable work to be done at the ordinary working distance. It is only when the patient will not be apt to change his glass after a year or two that we may occasionally prescribe in practice glasses that are a little stronger than are needed. We have to individualize in regard to this matter. In the second place *the strength of the presbyopic glass depends on the visual acuity of the eye to which it is fitted.* The poorer the vision the closer must the near point be brought by the glass, in order to obtain larger retinal images. Presbyopia is a symptom of age and old people not infrequently lose something of their visual acuity, even when their eyes are otherwise healthy.

Numerous researches have been made with regard to the behavior of the visual acuity in aged persons. Although there are many exceptions to the rule yet in general the acuteness of vision decreases as age increases, because the irregularities of refraction normally present in the lens become greater, and the opacities in the vitreous are apt to multiply with age. The pupillary conditions also play a part. The diffusion circles become smaller through the senile contraction of the pupil, but the aberration of diffraction increases. In addition to this, nutritive disturbances take place in the parts of the retina sensitive to light in consequence of senile changes, the existence of which cannot be positively denied. In the third place *the choice of the presbyopic glass depends on the refraction of the eye.* Weaker glasses must be prescribed in low degrees of myopia, stronger ones in hypermetropia. We frequently find pathological changes in the eyes of presbyopes. Sometimes they tell us that they no longer need to wear convex glasses because they are able once more to read without them. It has already been mentioned in the discussion of senile cataract that this symptom is due to the development of a lenticular myopia, which may be the precursor of a cataract. In other cases of actual cataract an aperture in the pupillary region may permit of vision as through a stenopæic slit, which allows of vision at a greater or less distance without any change in the dioptric apparatus. All of these conditions show that we need to proceed carefully in this functional test of the eyes. To render the work easier I append the following table, which gives the correcting lens for near work for normal emmetropic presbyopes.

Correcting Glasses for Near Work According to *Holtz*

Age	Strength of the Glasses in Dioptres	Age	Strength of the Glasses in Dioptres
40	0.75	65	3.25
45	1.50	70	3.50
50	2.00	75	3.50
55	2.50	80	3.50
60	3.00		

193. The Objective Determination of the Refraction by the Direct Method with the Ophthalmoscope and by Skiascopy

We have thus far used the test of the visual acuity to determine the refraction and have found that *if in this test an eye needs no glass and does not see as well with a convex lens it is emmetropic; if it sees just as well or better with a convex glass as without, it is hypermetropic; if it needs a concave glass it is myopic.* Such an examination is often very wearisome and it may take a long time to determine satisfactorily the refraction of an eye in this manner. For this reason objective methods of examination have been perfected which make us independent of the statements made by the patients and enable us to determine the refraction of an eye quickly and positively. The two most important of these methods are the measurement of the refraction with the ophthalmoscope in the upright image, and skiascopy.

I. Determination of the Refraction of the Eye by the Direct Method

In order to determine the refraction of an eye by the direct method, or in the upright image, we start from the known fact that the normal emmetropic eye is so constructed that, when the accommodation is at rest, the parallel rays which fall on the cornea are focussed so as to form a sharply defined image on the retina, and that, inversely, the rays coming from such an emmetropic eye emerge parallel to each other. Our ability to see the fundus distinctly and clearly when we use the method of the upright image in our ophthalmoscopic examinations depends upon these two facts. We focus the parallel rays that come from the eye under observation upon the retina of our own eye. The course of the rays is shown in this diagram.

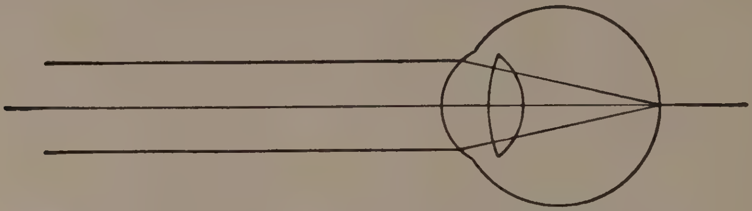


FIG. 166.—Course of the Rays of Light in an Emmetropic Eye.

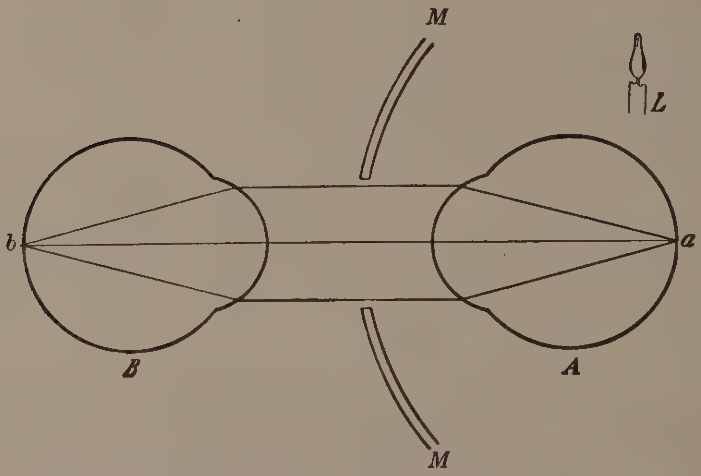


FIG. 167.—Course of the Rays of Light in an Examination with the Upright Image.

We assume, first, that the examiner has a normal, emmetropic eye, or that he has had it made emmetropic by correction. If we wish to examine a myopic eye in this manner we cannot a priori see the fundus distinctly, because the rays that emerge from the shortsighted eye are not parallel, but converge toward its far point, and the emmetropic eye of the examiner is not adjusted to focus such rays on its retina. Therefore in order to see distinctly the fundus of a myopic eye in this manner we must render parallel the convergent rays that emerge from it by interposing a concave lens in the ophthalmoscope which must be of exactly the same strength as the one needed to correct the myopia, for such a glass alone can render convergent rays parallel. If the eye that is being examined has, for example, a myopia of 4 D, parallel rays falling upon a lens of this strength take a direction as though they came from the far point of the eye, and inversely the rays emerging from the eye are rendered parallel by it. The fundus of the eye can then be seen distinctly, and it is evident at once that we can measure the degree of myopia in this manner. We interpose one concave glass after another, each stronger than the last, in the ophthalmoscope, and the weakest with which we can see the fundus distinctly gives us the degree of its myopia. Two points must be noted. The weakest glass is to be taken as the measure of the myopia, for the fundus can be seen with stronger ones when we accommodate and overcome the glass so far as it is too strong. The other point is that the macula lutea is the part of the fundus of which the refraction should be measured, but as this is usually not characterized by any special marks and is more difficult to recognize, we use for the purpose of measuring the refraction the temporal edge of the optic disc where we find a minute vessel coursing toward the macula. The refraction of the edge of the optic disc may differ a little from that of the macula. If the physician himself is nearsighted he needs a concave glass which corrects his own myopia plus the number of dioptres needed to correct the myopia of the patient's eye, in order to see the fundus distinctly. Suppose he has a myopia of 2 D and is just able to see the fundus clearly with − 6 D, then the myopia of the patient's eye is 4 D. But if the physician is hypermetropic he should wear his correcting convex glass when he is engaged in measuring refraction. If, for example, he has 2 D of hypermetropia and needs to use a − 6 D lens in his ophthalmoscope in order to see the fundus of a myope, the latter has a myopia of 8 D, for the convex glass correcting the hypermetropia of 2 D neutralizes the effect of a concave glass of 2 D. If the physician knows the exact amount of his hypermetropia he will not be obliged to wear his correcting lens, he will need only to add the amount of his hypermetropia to the concave glass found.

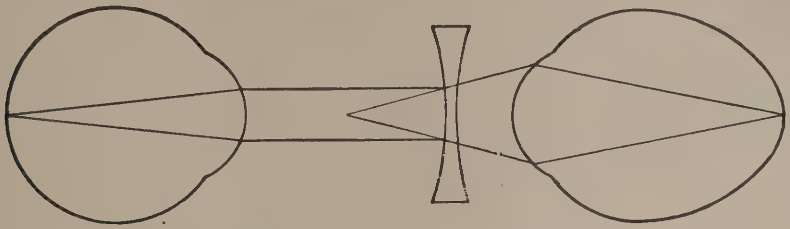


FIG. 168.—Measurement of Myopia with the Upright Image.

Measurement of Hypermetropia

The hypermetropic eye, when it does not accommodate, is adjusted only for such rays as fall upon the cornea with a certain degree of convergence. Inversely the rays emerge from such an eye with a certain degree of divergence, and in order to enable an emmetropic eye to focus such rays upon its retina it is necessary either that it should accommodate, or that they should be ren-

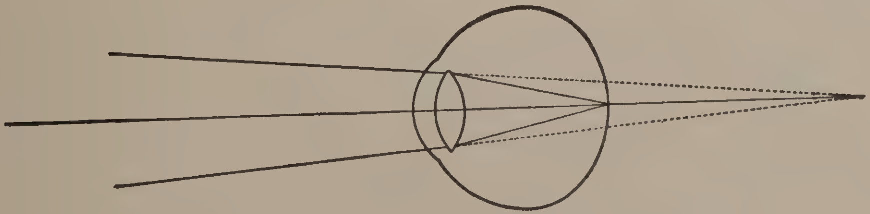


FIG. 169.—Course of the Rays of Light in Hypermetropia.

dered parallel. The latter is accomplished by the interposition of convex glasses, and the course of the rays in that case is shown by the diagram.

The convex glass which gives the rays that fall upon a hypermetropic eye a sufficient degree of convergence to enable them to be focussed on the retina corrects the hypermetropia, and the same glass renders parallel the divergent rays that emerge from a hypermetropic eye. The strongest convex glass with which the fundus can be seen distinctly gives us the measure of the hypermetropia of the eye. The advantage of this method is that we can measure the total hypermetropia in this way, while in the subjective examination a portion of the hypermetropia is concealed by the accommodation, or remains latent. If the physician himself is hypermetropic he must subtract the amount of his own hypermetropia in dioptres from the number of the strongest convex lens with which he can see the fundus distinctly. If he has a hypermetropia of 2 D and finds +6 D to be the strongest glass with which he can get a clear view of the fundus, the patient has a hypermetropia of 4 D. If he is myopic he needs a weaker convex lens with which to see the fundus than an emmetrope and must add the number of dioptres of his own myopia to the glass found. For example, if a myope of 2 D uses a convex glass of 2 D in order to see the fundus, the eye examined has a hypermetropia of 4 D.

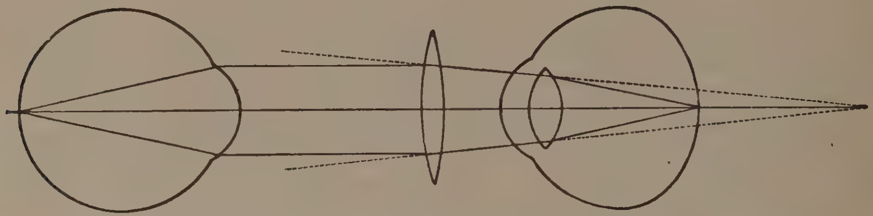


FIG. 170.—Measurement of Hypermetropia with the Upright Image.

II. Skiascopy

The second objective method of determining the refraction is the shadow test, or skiascopy, devised by *Cuignet*. We may illustrate the technique of this method of examination, without going into its theoretical portion, by the following experiment. Let the examiner sit in front of the patient and throw light into his eye from some distance with a concave mirror. If any parts of the fundus can be seen distinctly from this distance the eye is either highly hypermetropic, or highly myopic. The examiner can ascertain which of these conditions is present by two movements: he may either move his head from side to side, or he may move his head and mirror directly toward the eye. If the eye is highly myopic we know that the rays that emerge from it are convergent, that an inverted image of the fundus is formed at the place where they intersect, and that we can see this inverted image from a greater distance, so the examiner ascertains by his own movements whether what he sees is an inverted image, and so whether the eye is myopic or not. If he moves to the left the image of the fundus moves to the right, and vice versa. If he moves forward toward the eye the image grows more and more indistinct, because he very soon approaches so near to it that he can no longer accommodate for it. If, on the other hand, the eye is very hypermetropic he can see the details of the fundus distinctly from even a greater distance, but the image is upright, moves in the same direction as the examiner and remains distinct as he approaches the eye.

We can also determine accurately other degrees of hypermetropia and myopia, and this brings us to the true shadow test. If the examiner sits in front of the patient at the distance of 1 metre, throws light into the eye of the latter with a concave mirror of 20 cm focal distance, and then makes short rotating movements of the mirror about its vertical axis the illuminated field of the pupil seems to swing to and fro. By looking carefully it will be seen that the red light moves in the pupil and gives rise to a black shadow at the margin of the latter which wanders over the entire pupil as the mirror is rotated until it has become totally dark. The method of examination has received its name from this shadow. *We can determine the actual construction of the eye from the direction in which the illuminated field or the shadow moves with respect to the movements of the mirror.* We need only to divide all the conditions of refraction into two large groups, one including all the degrees of myopia from 1 D upward, the other myopia of less than 1 D, emmetropia and hypermetropia, and then both theory and practice will give the following results. The movement of a light reflected from the retina of the eye that is being examined is seen in the upright image as long as the far point of the eye is behind the eye of the observer, and in the inverted image when it is situated in front of his eye. Remember that all objects in space cast inverted images on the retina and that the movement of the light is likewise reversed on the retina. Hence if the shadow moves in the opposite direction to the movement of the light thrown into the eye the image is upright and the conclusion is that the far point of the eye undergoing examination is behind the eye of the observer, and that the eye itself is either hypermetropic, emmetropic, or myopic to a degree less than 1 D, for when the myopia is greater than this the far point of the eye is closer to it than 1 m. But if the light is thrown by a concave mirror of 20 cm focal distance from a distance of 120 cm and if the eye is shortsighted, the shadow will move in the same direction as the movement of the mirror. If the mirror is rotated to the left the shadow moves to the left. The rays emerge from the myopic eye in a convergent direction so as to intersect in front of the eye, and when this is the case the shadow moves in the same direction as the rotation of the concave mirror. Even when the myopia amounts to only 1 D the far point of the eye is situated at a distance of 1 m and the rays intersect before the face of the examiner. The conditions are reversed when the far point is situated behind the eye of the observer in low degrees of myopia, or behind the eye of the patient in hypermetropia. In these conditions the shadow moves in the opposite direction to the rotation of the concave mirror. Hence *a single rotation of the mirror is sufficient to tell us whether a myopia greater than 1 D, or a less degree of myopia, emmetropia, or hypermetropia is present.*

If the shadow moves from the start in the same direction as the rotation of the mirror there is present a myopia which is greater than 1 D, but if it moves in the opposite direction the eye is either slightly myopic, emmetropic or hypermetropic. Now we need only to interpose suitable glasses to neutralize the refractive error present until we find one which causes the shadow to reverse, or to move in the opposite direction, in order to ascertain the degree of ametropia. If the shadow moves in the same direction as the rotation of the mirror we interpose concave glasses, beginning with weak ones and changing them for stronger, before the eye, if it moves in the opposite direction, we use convex lenses in the same way. Various skiascopes have been made for the purpose of enabling the change of glasses to be made rapidly. We use the apparatus devised by *Hess* and proceed in the following manner.

We place the patient in front of the instrument, which consists of a rotatory disc, that contains convex glasses in one half, concave ones in the other, affixed to a rod. The eye to be examined looks forth at first between the two series of glasses. We cast light into the pupil with a concave mirror of 20 cm focal distance from a distance of 120 cm, make a rotating movement of the mirror a few times about its vertical axis and note whether the shadow moves with or against the movement of the mirror. If the movement is with the mirror the eye has a myopia of 1 D or more. The disc is then rotated so as to bring one concave glass after another before the eye until one is reached with which the shadow reverses and moves in the opposite direction. This glass has revealed the far point of the eye over 1 m (1 D). The myopia of the eye was corrected to within about 1 D by the last glass with which the movement of the shadow was with that of the mirror. Therefore the total myopia is found by adding 1 D to this glass. In this patient the movement of the shadow was with that of the mirror with a glass of – 3 D, therefore the myopia amounts to 4 D. Here is another patient in whom the shadow moves against the movement of the mirror from the first. He therefore has either a slight myopia, emmetropia or hypermetropia. Convex glasses are brought in front of his eye until the shadow reverses and moves with the mirror. Now we have to deduct 1 D from the number of this glass to find the true refraction. If the reverse takes place when we use +1 and we subtract 1 D we have emmetropia. If the shadow reverses with +3, we have a hypermetropia of 3 D – 1 D=2 D.

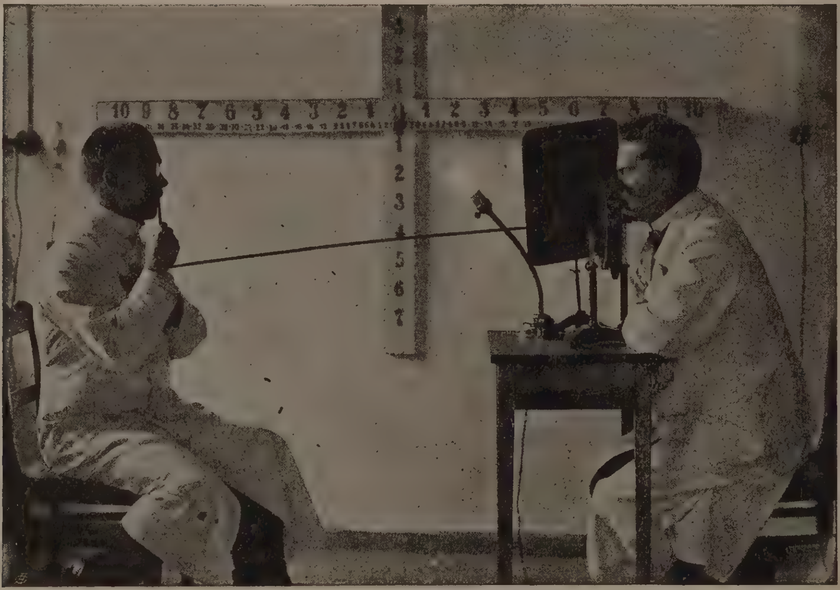


FIG. 171.—Skiascopy.

While the determination of the refraction with the ophthalmoscope requires greater experience and is frequently not pleasant to use, as when the patient has ozæna, skiascopy can be learned in a few minutes. One advantage of the latter is that we can easily determine the refraction in the principal meridians of the eye. We use the skiascope first in the horizontal meridian, then turn the mirror and investigate the vertical meridian, so that we are informed at once if any astigmatism is present, and this is certainly a great advantage.

As regards the accuracy of this method it must be borne in mind that we skiascope the region of the papilla, and that sometimes differences exist between the refraction of the papilla and that of the macula, which readily appear on the functional test. Otherwise the advantages of this method are so overwhelming that it is inconceivable to me that there should still be large eye clinics in which it is too little used. When one sees the time spent by physicians in such clinics in order to fit glasses by the subjective method he can only think it is because they do not know the value of skiascopy. With us the examination of every case of refraction is begun with skiascopy, except in the highest degrees of myopia. We are thus immediately informed concerning the construction of the eye. We use, when necessary, the ophthalmometer of *Javal*, and then we know at once about what glasses will be needed. The measurement of the refraction of the eye is thus made in a few minutes. The value of skiascopy is particularly appreciated by those who are engaged in the examination of the eyes of school children.[1]

194. The Myopic Eye

I. The External Appearance of the Myopic Eye

A myope sees indistinctly at a distance because of the presence of diffusion circles, so he tries to help himself by squeezing his lids together so as to form a sort of stenopæic slit and make the circles of diffusion smaller. The name myopia comes from this peculiar screwing up of the eyes. When we look carefully at the eye of this patient we find that its anterior segment does not differ from the normal, and yet it can readily be established that the form of the myopic is not the same as that of the emmetropic eye.

It is only necessary to direct a patient who has a high degree of myopia to look strongly inward to reveal that the segment of the sclera lying behind the equator is much stretched. The region of the equator does not bend backward in a large curve as in the normal eye, but runs backward without much of a curve. *The myopic is longer than the normal eye,* and in the overwhelming majority of cases the myopia is due to an elongation of the axis. The enlargement is frequently shown by the fact that the eyeball protrudes from between the lids.

A priori a myopia may be caused by too great a curvature of the cornea, or of one of the two surfaces of the lens, by too high an index of refraction of the cornea and aqueous, by too low an index of refraction of the vitreous, or by an abnormal nearness of the lens to the cornea, as after an escape of the aqueous, or in subluxation of the lens, but such cases are exceptional. The anterior chamber is apt to be rather deeper in myopia than in emmetropia. Tears in *Descemet's* membrane are occasionally to be seen in the higher degrees, but the principal changes take place in the posterior segment of the globe.

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[1] For skiascopy as practiced in this country see Appendix by the translator.

II. The Course of the Rays of Light in the Myopic Eye and the Correction of Myopia

The degree of myopia may be ascertained either objectively by an examination of the upright image with the ophthalmoscope and by skiascopy, or subjectively by the test of the vision. This patient is unable to read the largest test type at a distance without a glass, but has perfect visual acuity with a – 5 D lens. Near vision is excellent. Such rays of light only are focussed on the retina as enter the eye with a certain degree of divergence.

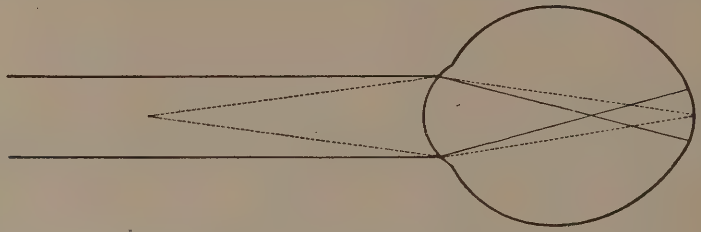

FIG. 172.—Course of the Rays of Light in Myopia.

The point to which the myopic eye is adjusted when it is in a state of rest is called the *far point*, and *is situated at a finite distance from the eye.* Beyond this point vision is indistinct without a glass. This patient is just able to read print 20 cm away, so his far point lies 20 cm in front of his eye. This method gives us a certain guide to the amount of myopia in the higher degrees. The problem we have to solve is how to enable the eye of the patient to focus parallel rays of light upon its retina and so enable him to see well at a distance. The solution is to place in front of the eye the concave glass that will give to the parallel rays that fall upon it the requisite amount of divergence which will cause them to follow a direction as though they came from its far point; the myopic eye can see distinctly at a distance as soon as the lens that will do this has been obtained.

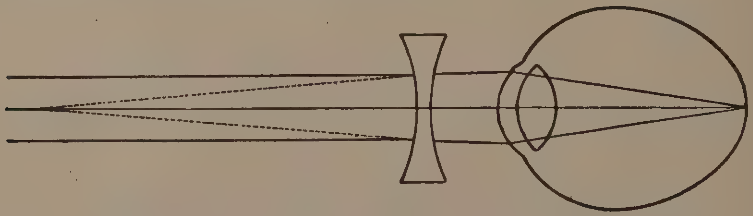

FIG. 173.—Correction of Myopia by Concave Glasses.

The myopic eye sees distinctly at a distance with that concave glass the focal distance of which is equal to the distance of the far point from the eye. Inversely, the focal distance of the concave glass with which the myope sees well at a distance gives us the degree of the myopia. In this case we need a concave glass of 20 cm focal distance, and the eye has a myopia of 5 D. There remains only one thing more, a few words of advice concerning the measurement of myopia by means of glasses. When a myope puts on stronger concave glasses than he really needs he can still see well at a distance with them as long as he is still young. The rays of light that enter his eye are more divergent than is necessary, but he overcomes to a certain degree the excess of strength of the glass by means of his accommodation. Hence it is easy to make the mistake of prescribing too strong glasses in myopia. Such an error forces the accommodation to undergo constantly an unnecessary strain. *The rule is that the weakest concave glass with which the best vision is obtained should always be prescribed for myopia.*

Aside from the trouble of bad distant vision myopes are apt to complain of a lack of endurance in near work, especially when the refractive error is of high degree. Owing to the short distance at which the far point lies a constantly increasing convergence of the visual axes is necessary, often the internal recti cannot perform their work continually and there is an *insufficiency of the interni* which gives rise to the troubles symptomatic of *muscular asthenopia.* Finally *strabismus divergens* is frequently associated with myopia. There is only one other point to which I wish to call attention. It often happens in myopia that normal vision cannot be obtained with the correcting glasses. The relations between visual acuity and myopia have been studied a great deal, and it appears from the researches that have been made that the visual acuity sinks as the myopia increases. This is a fact that is readily comprehensible when we take into consideration the changes that take place in the fundus.

III. The Ophthalmoscopic Changes in the Myopic Eye and Their Anatomical Causes

The so called *conus* is found very commonly on the temporal side of the optic nerve. This is a bright, white crescent which varies in breadth according to the degree of myopia, although no mathematical relations exist between its breadth and the degree. In one patient it is narrow and uniformly bright, sometimes speckled on the side toward the retina, brownish red, or yellowish red. In another patient it is almost as broad as the papilla. In still other cases it extends around the entire papilla, surrounding it like a ring; this we call an annular conus. Traces of the chorioidal vessels may be seen within it in the last class of cases. A narrow conus may be met with in emmetropic or hypermetropic eyes, but the diagnosis of myopia may be based with certainty on the presence of a large one. The nature of the myopic conus and the way in which it is brought about have long been subjects of energetic discussion. In order to understand the ophthalmoscopic picture it is necessary to study the microscopic sections, although it has been my experience that the translation of the changes seen with the microscope into what is seen ophthalmoscopically is always attended with much difficulty, because the microscopic changes appear to be insignificant and impress the observer very little in comparison with the imposing spectacle of the ophthalmoscopic picture, besides which we have only a minute portion of the tissue before us under the microscope. The pigment epithelium ceases in the vicinity of the optic nerve, especially at the site of the conus, and where this is drawn away firm adhesions are formed between the retina and the subjacent tissue. The conus must be supposed to be an imprint caused by a stretching of the posterior segment of the eyeball. The ophthalmoscopic picture is due to the fact that the pigment epithelium of the retina stops at some distance from the papilla and consequently leaves visible the parts situated behind where it should be. If the tissue of the chorioid is still present the conus looks yellowish, reddish, or brownish, but if this tissue is absent the conus is white, because the inner surface of the sclera, or the inner wall of the scleral canal, is visible. As we ascribe the conus to the stretching which the tissue undergoes in myopic eyes we consider it to be an acquired condition. Together with many other ophthalmologists I am of the opinion expressed by *Schnabel* and *Herrnheiser* that the occurrence of the myopic conus as a congenital anomaly has not yet been proved.

Another change with which we are apt to meet in myopia is the so called *supertraction*, in which the edge of the chorioid is drawn over the nasal margin of the optic disc.

The stretching to which the myopic eye is subjected is most marked at its posterior pole. To give an idea of what the retina has to sustain in such an eye I will say that according to *Hess* the retina covers a surface of about 9 square centimetres in the portion of the eye lying behind the equator. If the eye attains an axial length of 31 mm this surface must be increased by several square centimetres and we can conceive at once how the retina may suffer injury from such a stretching.

A peculiar ophthalmoscopic picture is produced when the posterior segment of the sclera is bulged out like a staphyloma, that is, when an *ectasia of the sclera* is present. We then find at the posterior pole usually only on the nasal side, peculiar shadings that look like dark crescents, at which the vessels of the retina are bent sharply. Centrally from these places the refraction is higher in degree, so that we can recognize from this the bulging outward of the sclera. The ophthalmoscopic picture seen has been aptly compared to the appearance of terraces.

Weiss has called attention to a lustrous "*reflex curved line*" which may appear on the inner side of the head of the optic nerve in commencing myopia. This phenomenon he explains as produced by an accumulation of fluid in front of the papilla.

The *changes at the posterior pole* of the eye are of far more importance. We may find *in the region of the macula large, bright, irregular patches*, the margins of which are much pigmented. This is frequently taken to be a chorioiditis, but it is primarily the result of mechanical injuries produced by the stretching. At least, dehiscences in the tissue of the chorioid and defects in the elastic lamina are often found in such eyes on anatomical examination. Sometimes we find in the *macula* a round, *black spot*. Such patients complain of metamorphopsia and of a rapid impairment of the vision; they may have distinct central scotomata. The trouble in these cases is mainly a great proliferation of the pigment epithelium. One danger to which persons with high myopia are subject is that *hemorrhages* are apt to take place in the *macula*, which gradually bring about other ophthalmoscopic changes and cause a considerable impairment of the vision. The most dangerous complication of high myopia is the *detachment of the retina*, which has already been discussed.

195. The Genesis of Axial Myopia

The first question that arises in connection with the genesis of myopia is whether it is ever congenital, even in the slightest degree, or is developed in the course of childhood and youth.

Many myopes remember that as children they saw just as well as their playmates without glasses, and we can deduce from this fact that myopia is developed. Other evidence is furnished from the examination of the eyes of infants. We find almost without exception that the eyes of infants are hypermetropic, if we first paralyze the accommodation with atropin. The hypermetropia, as based on statistics, averages from 1 to 3 dioptres. The compilation made by *Randal*, which included over 200,000 eyes, shows hypermetropia to be by far the prevailing refraction in childhood and the early years of youth. Statistics show further that outside of schools and among uncivilized peoples hypermetropia remains the prevailing form of refraction. Myopia is almost unknown in the earliest years, is rare in the early days of school life, becomes more frequent in the course of the school days, and reaches a deplorable height in the higher schools. A conception of the development of this condition of refraction in the human eye during life can be obtained by a glance at this table, which is formed from the statistics compiled by *Herrnheiser* from the examination of 11,000 eyes.

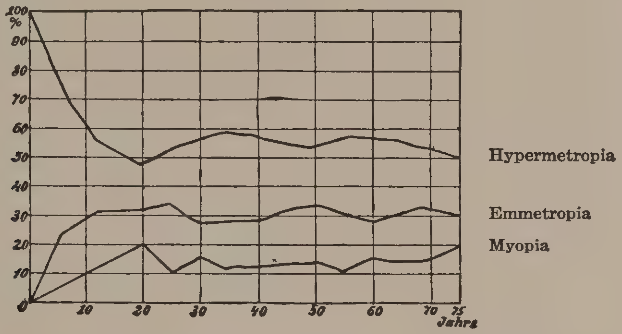

FIG. 174.—Table Showing the Development of Refraction during Life, after *Herrnheiser*.

From this table it can be seen that hypermetropia is the prevailing refraction even to extreme old age. Another fact of importance is that myopia increases slowly but surely up to the twentieth year.

We may conclude from all of these facts that myopia is not congenital, but develops during the period of the growth of the body. Hence the questions arise: Why do eyes become myopic during youth? What is the cause of myopia? We may make these questions rather more definite if we start from the fact that by far the commonest form of myopia is the axial and then ask: What forces operate in youth to bring about the great length of the axis of the eyeball, the elongation of the globe from before backward?

One factor has been known for three hundred years. The great astronomer *Kepler* said that young people who study a good deal are apt to become nearsighted. In fact the suggestion is obvious that the occupation of the eyes with the objects seen during near work is an essential factor in the production of myopia, and researches have been made for decades concerning this connection between near work and the elongation of the axis of the eye. The statistics that have been gathered from the investigation of more than 200,000 school children have resulted in the recognition of the fact that the number of myopes throughout the civilized world increases with the demands made upon the children by the schools and with the classes.

Various theories have been advanced to explain how near work exerts such an unfavorable influence in causing the development of myopia. Many ophthalmologists are of the opinion that *an increase of the intraocular tension takes place in the posterior segment of the eyeball during near work*, in the vitreous, which results in a stretching of the posterior wall of the globe, and so in a lengthening of the axis of the eye. This theory was voiced by *v. Arlt*, according to whom the change in the form of the eyeball is due to repeated temporary increases of the tension in the posterior part of the eye.

But how are these increases of intraocular tension brought about during near work? What proof have we of their existence? Under normal conditions two forms of muscular activity are called into play during near work with binocular fixation, accommodation and convergence. When I change from looking at a distance to fix upon the point of a pencil 20 cm away the axes of my eyes converge to this distance and I can see the object distinctly only when I focus my eyes upon it at the same time by means of my accommodation, and my pupils contract together with the contraction of the ciliary muscle which induces an increased curvature of the lens. If an increase of intraocular tension is produced by near work the only question is whether it is caused by the accommodation, or by the convergence, that is by the muscular activity inside or outside of the eye.

The theory was widely maintained for a long time that the accommodation furnished the force which produced a baneful effect in the genesis of near-sightedness and in its development, because an increase of tension in the eye is associated with it, and this theory has exerted a considerable influence upon the therapeutic measures that have been and are still employed in the treatment of myopia. Many versions of this hypothesis have appeared. Some ophthalmologists believe, on the ground of inconclusive experiments, that an increase of tension can be established during the process of accommodation, but *Hess* and *Heine* have proved by exact experiment that even the maximum contraction of the ciliary muscle can produce no increase of the intraocular tension.

Others believe that the posterior segment of the eye is pulled upon by the contraction of the ciliary muscle by means of its meridional fibres which lose themselves in the chorioid. Even if such a pulling by the meridional fibres of the ciliary muscle does take place, a glance at any section of the eye is sufficient to show that for physical reasons it cannot result in a stretching of the sclera, in a lengthening of the eye. Besides this *Hensen*, *Voelkers*, and *Hess* have shown that a slight movement of the chorioid can be observed at the equator and a little behind it during the contraction of the ciliary muscle. When fine needles are stuck into the eye in a proper manner they indicate the movement of the chorioid when the ciliary muscle is stimulated, but such movements are totally absent at the posterior pole, which is the main point with regard to this hypothesis.

Another version of the accommodation theory is that of *Mauthner*, that the pulling upon the chorioid may bring about mechanically a hyperæmia with increased exudation from the vessels. If this were true it is evident that the hyperæmia and transudation at the posterior pole would have to be much greater in hypermetropes than in myopes, because they accommodate a great deal more, but we do not find this postulate correct either in myopia or hypermetropia.

Some clinical objections to the accommodation theory may be added in case what has been said seems to be insufficient to prove that the hypothesis is untenable as an explanation of the supposed increase of tension during near work.

If it is the accommodation that causes the elongation of the axis of the eye through increased tension, why does myopia so often advance in a patient who has 4 D already, whose far point is as it were prepared for near work and who exercises no accommodation in performing it? Why does the sclera bulge if the contraction of the ciliary muscle increases the tension and the chorioid has to bear it all? If the contraction of the ciliary muscle causes the formation of a conus by the pulling exerted on the chorioid, why is the conus produced on the temporal side of the nerve when the meridional fibres contract uniformly and so should cause a circular atrophy of that membrane? Why is not the conus formed to the same degree in hypermetropes as in myopes, and why do the myopic changes in the fundus extend although the accommodation is no longer used in near work? This is sufficient. The mechanism of accommodation in the human eye can never bring about the elongation of the axis, or the changes in myopia by means of an increase of tension during near work. We need not credit Nature with such an inexpedient arrangement.

It has been suggested that a *pathological* condition of the ciliary muscle may be of influence upon the development of myopia. Now and then children come to the clinic who take stronger glasses in the subjective than are indicated in the objective examination. We also see the refraction recede somewhat in myopes after atropin has been placed in their eyes. Such symptoms have been ascribed to a spasm of the ciliary muscle. I agree with *Hess* that a true spasm of the accommodation is much more rare in myopia than has commonly been thought. For this reason it cannot explain the origin of myopia. Even if it were more frequent than it is such a myopia could not be caused by an accommodative increase of tension, because such does not exist. The entire question as to the causal connection between near work and the elongation of the axis of the eye rests upon the theory that during near work an increase of tension takes place in the posterior segment of the eye, but the one muscular activity in the eye during near work, the accommodation, cannot induce an increase of tension. There remains another muscular activity, which is exerted not in but on the eye during near work, and is, in fact, able to increase the tension in its posterior segment. This we know best from experimental researches which prove that the contractions of the extrinsic muscles of the eye may result in an increase of the intraocular tension which is not insignificant.

The overwhelming majority of ophthalmologists are of the opinion that these increases of the intraocular tension that take place during near work are due to *a direct compression of the eye* by the contracted muscles, but investigators are not agreed as to the part played by the individual muscles. Some ascribe the compression to the associated action of the externus and internus; others, like *Stilling*, hold the superior oblique to be responsible; still others, like *Donders*, avail themselves of the aid of an over distention of the eye with blood, which is due to the inclined position of the head, to explain the increase of tension during near work. Many conceive that such conditions of congestion lead to a softening of the tissue, when the normal tension of the eye, and still more an increased one, may bring about the stretching at the posterior pole. *Arlt* believes that during convergence certain muscles, particularly the external rectus and the inferior oblique, press upon the vasa vorticosa and hinder the outflow of blood from the eye, but in that case we should be able to find signs of dilatation and engorgement in those veins on anatomical examination, and such signs have not been found by *Fuchs*. So the only thing that can be said is that contractions of the extrinsic muscles of the eye do bring about increases of the intraocular tension which are by no means inconsiderable. Such contractions of the extrinsic muscles take place continuously during near work, for it is with their help that the movements of convergence are performed, that the eyes are depressed toward the book, etc. If we wish to refer the undeniable connection between near work and the elongation of the eyeball which is demonstrable from statistics simply to these increases of the tension associated with near work, we are obliged to suppose that the continuous compressions of the eyeball result in a stretching of the sclera and so cause an elongation of the axis. The best attempt to explain why the posterior segment of the myopic eyeball should be stretched by this muscular activity exerted upon the eye has been given recently by *Halben* in a skillful analysis of the problem. He says that the internal tension can "exert a distending action upon the sclera only at such places as those in which the equilibrium is not maintained on its outer surface by an equal or greater pressure in the opposite direction." "The only large surface of the sclera free from external pressure is its posterior hemisphere, and therefore this has to bear a great burden from the internal pressure." He thinks it is due to this mechanical factor that in myopia the eyeball approaches the shape of an egg and is not enlarged spherically in all diameters as in hydrophthalmos. The case in myopia is rather one of a growth of the eyeball under increased pressure, than of a stretching of what is already grown. But the result of such effects of pressure evidently depends not alone on the muscular activity, but quite as much on the constitution of the compressed organ, the sclera. Important preliminary matters must therefore be settled by attention directed to the sclera itself.

Thus far we have referred the connection between near work and the elongation of the eyeball essentially to the increases of tension that accompany the former, but it cannot be emphasized too strongly that this is only one factor taken from the relations between near work and myopia, and that it is by no means the only one. If the intraocular increase of pressure caused by the contraction of the extrinsic muscles represents the measuring factor in the relations between near work and myopia it may not be incorrect to expect that eyes in which myopia is in process of development should exhibit an increased tension, but it must be admitted that the question as to the condition of the intraocular tension has met with very different answers although one would suppose it to have been definitely settled long ago. Some ophthalmologists believe the tension of myopic eyes to be increased, especially when the myopia is of high degree, others do not think that it is. I myself do not believe the tension of a myopic eye to be high. Here the advocates of the theory that refers the influence of near work to the increase of tension alone encounter a difficulty. When the tension is permanently increased in a young eye, as in the glaucoma of childhood, called hydrophthalmos, quite different changes are brought about than are met with in myopia. Portions of the anterior segment of the eye, or all parts of the globe, become enlarged, while in myopia it is the posterior segment alone that undergoes distention. The reply to this may be that there is a continuous, powerful increase of tension in glaucoma, while in myopia the increase is only temporary and feeble. The statement also has been made that in myopia the external pressure of the muscles is opposed to the internal pressure in the anterior segment of the eye, and that in hydrophthalmos the increased internal pressure originates and acts when the external pressure force is not acting. Whether these factors suffice to cause such essential differences as exist between myopia and hydrophthalmos is a question that is still open for discussion. Near work is acknowledged to be an important factor in the pathogenesis of myopia. We also admit that near work brings about increases of the intraocular tension through the action of the extrinsic muscles, but it is doubtful whether we have exhausted all the relations between near work and the elongation of the eyeball when we have dealt with these increases of tension. There are two clinical facts which the advocates of this theory are unable to explain. They cannot tell why it is that the most yielding portion of the fundus, the lamina, is not impressed with the optic nerve by the continually recurring increases of tension produced by the contractions of the muscles, as it is in the typical picture presented by glaucoma, and that only the vicinity of that tissue yields to the pressure. No version of the convergence theory can explain the second clinical fact that a person who has but one eye, or has had from childhood one eye so very myopic that it could never be used together with the other, and therefore cannot converge, may become myopic in the good eye.

Let us go a step farther and inquire whether near work, which is certainly a cause of myopia, is the only cause.

We have all been obliged to accommodate and converge from childhood, the yoke of near work, and we may even say of unnecessary near work, has been borne by us all in school, and yet only a few among us have become nearsighted. We ophthalmologists meet with many men who are very myopic and yet have not been tormented with near work in their lives. We know that in many countries whole families of peasants, the members of which can neither read nor write, are found suffering from progressive myopia, and it is difficult to maintain that near work has caused the myopia in such cases. Another point. We very often find that one eye of a patient remains perfectly normal, while in the other, which is amblyopic, strabismic, and never used for near work, the myopia steadily progresses. From these three facts, that in spite of near work only a percentage of mankind becomes nearsighted, that nearsightedness may develop without near work, and that it may affect only one eye, it follows that near work cannot be the only cause of the elongation of the eyeball. I therefore make the assertion that near work alone and of itself does not make man myopic. Either there are other causes that operate to elongate the axis of the eye, or something must cooperate to further the harmful effects of near work. When the true cause of a disease is unknown to us the first attempt of science is to explain its genesis from the supposed cause with the assistance of predisposition, a term under which is grouped together everything that favors the origin of a disease. Many attempts have therefore been made in ophthalmology to investigate the factors which predispose to myopia, all of which may be divided into two large categories for the sake of clearness.

Some ophthalmologists have sought to ascribe an influence upon the development of myopia to the parts *outside the eye*, especially to those in its immediate vicinity; others have looked for the favoring factors *in the eye itself*. Certain *relations between the form of the skull* and myopia have been sought for, and it has been claimed by *Mannhardt* that a form of the skull which necessitates a great distance between the points of rotation of the eyes must favor the development of myopia, because the points of rotation of the eyes are situated farther apart when the skull is broad, and when the eyes make a movement of convergence from such a distance the external rectus must compress the eyeball more than it does when the skull is narrow. Hence it must induce an increase of tension and an elongation of the eyeball. According to this a broad skull predisposes to myopia, a narrow one to hypermetropia. But what is actually the case as regards the pupillary distances in myopia, particularly during the years of its development and that of the organism? The answers that have been given to this question are extremely contradictory, but the more carefully measurements have been made the more certainly has it been established that a causal connection between the refraction and the pupillary distance, which could explain the origin of myopia, does not exist. Therefore the external form of the skull cannot be held responsible for the development of myopia.

Another attempt to furnish an explanation was made by *Hasner*, who believed that *the trunk of the optic nerve was absolutely or relatively too short* in myopes. If this were the case the optic nerve would exert a traction upon the sclera in the region of the posterior pole to the outer side of the nerve during movements of convergence, and so might give rise to the myopic conus on the temporal side of the papilla. He assumed erroneously that the conus was a bulging outward of the sclera. This hypothesis was later amplified and perfected by *Weiss*, according to whom the portion of the optic nerve that forms its S shaped bend is the difference between its length and the distance from the optic foramen to the posterior surface of the sclera. If the S shaped portion happens to be small he claims that signs of traction appear in the posterior segment of the globe. So much for the hypothesis. What are the facts in regard to the length of the optic nerve in myopes? In 200 autopsies *Stilling* found an abnormally short nerve very rarely, and in the cases in which such a nerve was found the eyes were normal. The anatomical examination of the myopic eye interposes the objection, as *Hess* has rightly said, that a traction on the part of the optic nerve in no way explains the uniform distention of the entire posterior segment of the globe back of the equator, which is frequently to be found. We must therefore consider this attempt to explain the predisposition to myopia by the topographical conditions of the length of the optic nerve as a failure.

The same is true of *Stilling's* hypothesis concerning *the connection between myopia and the construction of the orbit.* Starting from the assumption that occupational myopia is brought about by the pressure of the extrinsic muscles upon the eye he investigated the positions of the individual muscles relative to the sclera. As the direction of the entire muscle pressure is determined by the course of the superior oblique as the only variable factor *Stilling* believed that the predisposition to myopia depended on whether the tendon of this muscle was or was not able to exert a pressure upon the eyeball. The superior oblique arises in the orbit near the optic foramen, passes forward to the nasal end of the upper orbital margin, loops itself about the trochlea and then passes backward to the eyeball. He maintained that if the trochlea lies low in the orbit the tendon of the muscle lies in such a way on the wall of the globe as to exert pressure upon it when the eye is directed downward and so may lead to an elongation of the axis. According to *Stilling* this rotator of the eye may exert such a traction upon the optic nerve as to change the form of the papilla, which is usually drawn outward so as to render visible a portion of the scleral funnel, and this is the myopic crescent. The process is started by the little twitching movements that rapidly follow each other during near work. On the other hand, if the situation of the trochlea is high the tendon of the superior oblique cannot press so firmly upon the eye and compression is not produced. The position of the trochlea depends, according to *Stilling*, on the height of the orbit, which should be broad and low in a myope, while the orbits of emmetropes and hypermetropes should be narrower and higher. *Stilling* established an orbital index for the purpose of comparison, the ratio of the breadth to the height of the orbit, expressed in figures. He found the index in myopes to be 78, in emmetropes and hypermetropes 89, and therefore believed myopes to have lower orbits than emmetropes or hypermetropes. The origin of myopia might be a question of race, a high orbit might be a precondition of emmetropia and hypermetropia, a low one of myopia.

Subsequent investigation failed to confirm *Stilling's* theory. Direct measurements of the orbital index sometimes give the exactly opposite results, myopes very often have higher orbits than emmetropes, and above all the theory was found not to apply in anisometropia. If one eye is hypermetropic or emmetropic and the other myopic, the myopic eye should have the lower orbit, but in such cases the orbital indices are often the same, and sometimes the orbit of the myopic eye is higher than the other. The suggestion that the development of myopia may be a question of race has also been disproved. *Stilling's* claim that the form of the orbit exerts an influence on the form of the eyeball has not been proved. On the contrary our experience goes to show that the form of the eyeball influences that of the growing orbit. We know that the orbit remains backward in development after enucleation of an eye during childhood. This has been demonstrated experimentally by *Thomsen*. It has also been proved in man by *Schmidt-Rimpler* and *Seggel* that the height of the orbit increases with the enlargement of the eyeball.

None of these hypotheses which would connect the distention of the posterior segment of the eyeball with the arrangement of the parts surrounding the eye are able to explain the development of myopia.

Since near work alone does not suffice to produce myopia through its influence on the intraocular tension the only thing that remains is that one or more causes exist in the eye itself to account for the elongation of its axis. If the stretching of the sclera is not due to near work alone, nor to the arrangement of the parts surrounding the eye, nor to the action of the muscles, nor to optical conditions, its cause must be located in the sclera itself. I am one of the ophthalmologists who believe that *the cause of the elongation of the axis of the eye in myopia is to be found in an abnormally yielding nature of the posterior segment of the sclera.*

There are two ways in which this abnormal extensibility of the sclera may be conceived to originate. It may be hereditary, as suggested by *Schnabel*, in which case the congenital anomaly simply undergoes further development, or it may be an acquired property, and in that case it must represent a disturbance of the metabolism in the youthful sclera and of its relations to the general metabolism of the youthful, growing organism. It has been well said that the stretching of the sclera can take place more easily in proportion as that tissue is thinner and softer from the time of birth, and that great differences in this respect are to be met with. A number of investigators consider the most important etiological factor, the primary cause of the myopia indeed, to be a *congenital impairment of the power of resistance of the sclera.*

Schnabel and *Herrnheiser* thought they were able to demonstrate a congenital fault of development in the outer membrane of all myopic eyes, which consisted of an unusual softness of the sclera about the optic nerve, of a weakly development of the outer layers of the sclera and of an abnormal width of the sclerotico chorioidal canal.

What part does *heredity* play in the *genesis of myopia?* We know that myopia is not congenital, that the eye of the newly born infant is hypermetropic. Heredity can therefore furnish only a predisposition to myopia, a tendency of the eye to gradually elongate. But this very circumstance renders it extremely difficult to pass a positive opinion concerning the part taken by heredity in the genesis of myopia. It is certainly possible that the form of the eyeball may be transmitted to posterity, just as we often see attributes, features, and physiological conditions transmitted in a wonderful manner by parents to children, and every ophthalmologist knows of families in which the myopia may depend largely on heredity. But there are two things that must not be forgotten. One is that in comparison with the entire number of diseases of the eye those that are known to be hereditary are very rare, while myopia is very common. The other is that we have no sure criterion for a hereditary myopia. When we meet with a family in which the father or mother and one or more children are myopic we do not know whether the myopia of the children is not due to the same causes that produce it in those who have parents whose eyes are normal. The myopia of the parents need not have been inherited, they may have acquired it as children at school under the influence of near work. Investigation has shown that children of parents with normal vision who belong to the lower classes are apt to become nearsighted when they attend higher schools than those that were attended by the parents, and the uncritical statistics, according to which over 50% of the myopes were burdened with a hereditary taint, have long since been given up. In order to obtain some degree of clearness as to the part played by heredity in the origin of myopia we must begin by comparing families with hereditary taint with others that are without. *Pflueger* has furnished us with statistics of this nature. Of 100 families without hereditary trouble 26 had myopic children, 74 did not. Of the children belonging to these families 8.4% were myopic, 91.6% had other conditions of refraction. Of 100 families in which either the father, the mother, or both were nearsighted 45 had myopic children attending the same schools as the others, 55 did not. Of the children of these families 19% were myopic, 81% had other conditions of refraction. The percentage of myopes was greater in both classes of families in the higher schools. Forty per cent. of the families without hereditary taint and 71% of those in which heredity was present had myopic children. Seventeen of the children of the former, and 26% of the children of the latter were myopic. These results may be tabulated in the following manner.

Children Are Myopic

	I. Families with Hereditary Taint	II. Families with Healthy Eyes
(a) Lower Schools	19%	8%
(b) Higher Schools	26%	17%

This shows that *the influence of heredity may be assumed to be present in at most* 10% *of all cases of myopia.* The figures given by other investigators are larger; *Tscherning* found 14%, *Kirchner* 15%, and *Schneller* up to 25%. The last is too high a figure, in my opinion.

Laqueur suggested that the malignant form of myopia might be connected with inbreeding, and that consanguinity exercises a harmful influence in this respect. In the statistics furnished by *Wolff* consanguinity is given as the etiological factor in 8% of the cases of malignant myopia, but a sharp line cannot be drawn between the malignant and the nonmalignant forms, and this alleged harmful effect of consanguinity has not been confirmed by other statistical investigators.

Attention was early drawn to the fact that the highest degrees of myopia are more common in women than in men. Although the number of myopes is greater among males than among females we meet with malignant myopia, with disease of the macula, according to my observation, more often in the latter than in the former. To recapitulate the results of this survey of the part played by heredity in the production of myopia we may say: The abnormal extensibility and stretching of the sclera is due to a congenital weakness of the tissue only in a small proportion of myopes. If this lack of firmness of the sclera can be ascribed to heredity with some degree of probability in only between 10 and 20%, the remaining 80 or 90% must acquire it in the course of life, and it is by no means necessary that the rudimentary condition of the outer layers of the sclera and the weakness of this membrane in the vicinity of the optic nerve should be due to a congenital fault of development. I imagine that there is a sort of scleromalacia, analogous to osteomalacia, which develops in some persons during the period of their growth, recovers spontaneously in many cases, leaves behind an elongation of the axis of the eye, but lasts for decades in the malignant ones, and occurs sometimes in one eye alone. I regard this scleromalacia as a metabolic disease of the eye, and consider it to be the real primary cause of myopia. I believe that disturbances take place in the afflux and appropriation of those substances which the sclera needs for its normal firmness and elasticity in this disease of the growing organism, but the details of what takes place in the organism and in the sclera, what disturbances of the cell activity lead to this extensibility of the posterior segment of the sclera, are altogether beyond my knowledge. The real nature of the genesis of myopia is still unknown.

196. Prevention and Treatment of Myopia

The theory I have advanced is that in a certain percentage of mankind an abnormal weakness of the posterior portion of the sclera results from disturbances in the metabolism of that membrane and of the entire organism during the period of growth, and that this may be looked upon as the true primary cause of the development of myopia. We have also learned that *external* causes cooperate with this hypothetical *internal* cause, especially the action of the muscles in near work. It has been calculated that about 8% of the population is nearsighted. In that case there are 4,800,000 myopes to be found among the sixty millions of the German people. Even if these figures are too large the number remains frightfully too great. Are we completely impotent in this matter?

Before we attack the problem of the treatment of myopia we need to know whether all of the various degrees, from the lowest to the highest and most malignant, are simply degrees of one and the same disease, or whether there may not be various kinds of myopia. This question has to be studied from two points of view, the purely clinical and the etiological.

If we regard the different cases of myopia from the purely clinical standpoint we may divide them, like *Donders*, into three different forms: the stationary, the temporarily progressive, and the permanently progressive varieties.

Stationary myopia includes all of the lowest degrees in which throughout life an increase does not occur at all, or only to a very slight extent.

In the temporarily progressive myopia we see the nearsightedness increase considerably during a certain period of life, especially when the growth of the body is the greatest, up to 9 or 10 dioptres and then remain constant.

The permanently progressive myopia is characterized by the fact that throughout life, but especially after the growth of the body has been completed, an increase takes place up to the third or fourth decade and the changes in the fundus grow worse. These forms of myopia merge into one another and the question is: Can the second and third develop from the first?

One kind of myopia must first be excluded. We have been speaking thus far of axial myopia and have proceeded on the supposition that the nearsightedness was due simply to an elongation of the axis caused by a stretching of the sclera, but an eye may also be myopic when the proportion between the radius of the cornea and the axial length is not correct. The radius of the cornea and the axial length may vary within certain limits even in emmetropic eyes. A smaller radius of the cornea with a short axis of the eye, or a greater radius with a longer axis are consonant with emmetropia, but when a relatively small corneal radius coexists with a relatively long optic axis the result must be myopia. It has been calculated that with the axial length remaining the same a change of 1 mm in the radius of the cornea causes a change of the refraction amounting to almost 6 dioptres. Such cases, in which the nearsightedness is due to a disproportion between the focal distance of the dioptric system and the length of the axis of the eye, are known as *myopia in a perfectly healthy eye*, but they are rare. Having excluded this variety we will return to the question whether the different forms of axial myopia are only different degrees of one and the same disease, or are different diseases. It is evident that the clinical division of myopia given above needs to be supplemented by etiological research.

The attempt has been made repeatedly in the last decade to differentiate several kinds of myopia. Thus a difference of kind has been held to exist between the medium and high grades. High myopia in particular has been distinguished by many from the milder cases as a malignant disease. The milder forms have been ascribed not to a true disease, but to a remodelling of an otherwise healthy eye during growth by the pressure of the muscles in a way that is to a certain degree physiological. With many other ophthalmologists I do not consider such a differentiation to be justified. It is a clinical fact that myopia may develop not only in childhood, but in later years, when growth has been almost completed.

It is not true that serious complications appear only in the "malignant" form of myopia. Every high myopia may be productive of such complications. Detachment of the retina occurs in cases that have been looked upon for years as ordinary occupational myopia. An arbitrary limit of a certain number of dioptres, below which the eyes are exempt from complications, cannot be established from clinical experience. Attention has also been rightly called to the constant connection between the changes to be seen with the ophthalmoscope in the fundus, that may be traced from the low to the highest degrees of myopia, all of which are plainly based on the common factor of the stretching of the membranes of the eye. We have, in my opinion, no tenable ground on which to deny a common, uniform etiology of these changes, and accordingly I agree with those who believe axial myopia to be a single disease which presents differences only of degree.

I am convinced that the different degrees of this disease are to be explained by the varying intensity and duration of the scleromalacia, which I believe to be the primary cause of myopia. As in other metabolic diseases of youth, e.g., rhachitis, there are different degrees of intensity in the abnormal distensibility of the posterior segment of the sclera, and in like manner a rapid, or a slow spontaneous recovery may take place. When this anomaly of the metabolism is slight and recovers after a short time, it must end in a low or a medium degree of myopia, but when it is greater, and especially when it is persistent, a greater and more marked distention of the sclera must be the result. It is also quite conceivable biologically that such a condition of the sclera may be developed on only one side, and that its most posterior segment may be the portion particularly to be affected, because its nutritive conditions and its relations to the neighboring tissues are different from those of the other parts.

It is plain that in most cases the organism is able to overcome this disturbance in the nutrition of the sclera and that when the latter regains a firmness and elasticity adapted to the conditions of muscle pressure the myopia must remain constant. It is also quite conceivable that such a nutritive disturbance may last for decades, that the sclera may not become adapted to these conditions, and that then the myopia will be permanently progressive for a long time.

It is obvious that deleterious influences, such as near work, with its continuous compression of the globe and increases of tension, can favor the development of myopia in such a faulty condition of the nutrition of the sclera. At the same time the development and progression of myopia with such a scleromalacia is perfectly understandable even without a great deal of near work, for if the disease is well marked a progressive myopia may develop in people who never perform any straining near work, as the use of the eyes for near vision is not absent in such persons.

We can obtain a natural explanation of all the clinical symptoms by means of this hypothesis of the primary cause of axial myopia, and we can also explain by it all of the therapeutic results that have been attained thus far in the treatment of myopia and the endeavors to put a stop to its extension. If the cause is to be found in a disturbance of the nutrition of the sclera which results in a stretching of the latter under the influence of unfavorable hygienic conditions, especially near work, our therapeutic duty is clearly indicated. All of our thoughts and efforts must be directed in the future to find ways and means, if possible, to arrest the abnormal yielding of the sclera at the beginning of an axial myopia, and to bring it back to normal. It is one of the duties of internal therapy to investigate this mystery of the metabolism. Whoever can succeed in freeing mankind from myopia will win for himself the laurels of immortality. No means are known at present wherewith this can be done, but in order to leave nothing untried we advise that particular care be bestowed on the myopic child whenever possible.

As there is no etiological therapy of myopia we ophthalmologists have to admit that we possess no means by which we can with certainty prevent the progress of myopia in an individual case. We can never promise to arrest a myopia. We can see certain signs ophthalmoscopically which indicate to us either that the advance is very slow, or has ceased, but when the myopia stops the arrest is due to nothing that we have done, but to spontaneous recovery. We can do nothing more than to eliminate the deleterious influences which favor the progress of myopia, but in doing this we secure many happy results.

The most efficient incubator for the development of axial myopia is the school with its compulsory near work, hence the fight against this condition must begin in the school and in the near work at home. In fact the attention of physicians, teachers and school authorities has been turned to the solution of this problem for more than fifty years, and the reformation of the external and internal methods of instruction is well under way.

School myopia is an important disease for two reasons: First, because we know that in a large percentage of cases this myopia is of a serious character, reaches a high degree, and induces diseases of the retina and chorioid. Even when such serious complications do not appear statistics show that the average vision declines with the increasing grade of the myopia. Occasional cases of from 7 to 12 dioptres are to be met with which have excellent vision with the proper correcting glass, but the visual acuity is impaired, as a rule. This is shown by the following table, compiled by *Seggel*.

Table of Seggel

DECREASE OF VISUAL ACUITY WITH THE INCREASE OF MYOPIA

Eye	Myopia	Average Visual Acuity	Eyes	Myopia	Average Visual Acuity
186	0.25	1.1	173	5.0— 5.75	0.65
74	0.5—0.75	0.92	103	6.0— 6.75	0.59
267	1.0—1.75	0.80	85	7.0— 8.0	0.55
239	2.0—2.75	0.77	68	8.0—10.0	0.53
186	3.0—3.75	0.75	26	10.0—13.0	0.40
200	4.0—4.75	0.73	12	14.0—20.0	0.13

These dangers of school myopia have not always been estimated from such a point of view. From an erroneous conception of the theory of evolution some ophthalmologists have imagined occupational myopia to be a suitable adjustment of the human eye to the conditions of culture, to be an improvement of the eye, but fortunately the time is past when such an idea gained credence.

During the first two-thirds of his life the emmetrope can do his near work easily by a slight muscular activity that gives him no trouble, he can see distinctly at all distances, and under normal conditions no myopia develops after the forty-fifth year. Occupational myopia is not a suitable adjustment of the eye to its environment. I am positively convinced that myopia must be fought as such, because it brings serious dangers to not a few of its victims and an impairment of the visual acuity to many. This brings us to the subject of school hygiene.

The first problem of school hygiene is the *construction of school desks and seats so as to maintain an upright position of the body.* All points that pertain to the proper seating of children are present in the form devised by *v. Esmarch.* "School children become round shouldered and nearsighted because they sit in a bad posture on bad seats. They grow crooked with sitting when the seat is too far from the desk, when it is too low in proportion to the desk, and when it has no suitable back. Therefore the school seat is not harmful only when the child is obliged to sit upright on it when reading or writing and can sit on it in this position for a long time without weariness. In order to attain this it is necessary that 1, the seat must be at the same distance from the floor, or footboard, as the leg of the child is long, measured from the popliteal space to the sole of the foot; 2, the seat must be as broad as the thigh is long, measured from the popliteal space to the back; 3, the rounded anterior edge of the seat must extend two or three centimetres farther forward than the inner edge of the desk; 4, the seat must be high enough to enable the child to lay his forearm conveniently on the surface of the desk when he is writing, without elevating his shoulders or depressing his head and back; 5, the lower part of his back must be adequately supported while he is reading. As these conditions change with the growth of the child the seats should be changed according to measurements at least every half year."

Physicians have ample opportunity to introduce a suitable arrangement of the place for working at home. It is not every child that can have such a hygienic study desk at home, and yet the eye of the poor child is of even greater value than the eye of the rich. A correct seat can be arranged at any table by drawing the chair near to it and regulating its height by means of a cushion or a blanket.

When children have a serious tendency to sit in a distorted position we sometimes employ devices for holding the body upright, or reading supports which prevent the drooping forward of the head, such as the horizontal reading support devised by *Duerr*, which, with a little modification, can be attached to a table.

The second thing in the programme for the fight against myopia is the *demand for proper light in the work room.* The visual acuity is intimately related to the illumination and sinks considerably as the light decreases. In the dark we have to approach objects very closely in order to recognize them. The baneful effect of near work in badly lighted rooms is admitted. An illumination of ten metre candles is the minimum for any room in which such work is to be performed. When the illumination is artificial the light must not be dazzling, must not be hot, and must not flicker.

Equally important are the reforms that have taken place in the methods of instruction and of school management. An example of each may be given.

1. Vertical writing should be recommended as strongly as possible. According to the researches of *Schubert* this is the only style of writing which not only favors, but actually necessitates an upright position of the body without lateral curvature.

2. The fight against school myopia cannot be successfully prosecuted without the constant cooperation of the expert physician, and therefore the increasing number of places that are being created for school physicians is a further advance.

The results that have been obtained thus far cannot be said to be satisfactory. There are only a few ophthalmologists who believe that an actual decrease in the percentage of myopia in the schools has been brought about by the hygienic measures that have been adopted. *Seggel* found in the course of fifteen years a diminution of myopia from 45% to 20.3%, and *v. Hippel* likewise believes that the frequency of myopia has been diminished by these means, but I am obliged to admit with most ophthalmologists that no great decrease of myopia has as yet taken place.

Such a result is not a matter of surprise to anyone who agrees that we have not as yet an etiological therapy for myopia, that we do not know and cannot influence the true nature of the abnormal yielding of the posterior segment of the eyeball. Our hygienic measures can only mitigate to some degree other factors that are productive of damage, they cannot do away with the fundamental cause of myopia. Thus it happens that some ophthalmologists, like *Schnabel* and *Herrnheiser*, think little of these measures of school hygiene, but they are wrong in this. It is certain that our efforts will not cause myopia to disappear, but the hope is well founded, and has already been partially fulfilled, that by the consistent carrying out of the programme the average degree of nearsightedness will be lowered somewhat by the diminution of the harm producing factors. This, of itself, is a great gain, for we know from careful investigations that under the influence of near work myopia is apt to increase and serious complications to take place within the eye. It is not so very rare for detachment of the retina to take place in eyes that have 9 D or less of myopia. If it is possible even to lessen somewhat the degree of nearsightedness of the growing youth we have sufficient reason to exert our powers to the utmost in favor of school hygiene.

What advice should the physician give the patient in an individual case of myopia? To begin with, a suitable pair of glasses should be prescribed which will enable him to see better. Here we find that the theoretical views concerning the mode of origin of myopia cut deeply into the practical life of the myope. Two courses are opposed to each other in the question of what glass to prescribe, although the antagonism between them is not so great as it formerly was. Whoever believes that the accommodation produces a harmful effect upon the disease of myopia must prescribe logically as weak glasses as possible and allow the patients to do their near work without a glass. It is not long since atropin was prescribed in the treatment of myopia, that the accommodation was carefully excluded for weeks and months by the paralysis thus induced of the ciliary muscles, and even today there are ophthalmologists who think that the accommodative effort should be at least limited as much as possible in myopia.

On the other hand all those ophthalmologists who believe that the contraction of the extrinsic muscles of the eye, especially in the convergence of the visual axes, is the harmful factor in the development of myopia prescribe the strongest possible glasses for near work with which the patient can read and write at the greatest distance and needs least to converge. Which standpoint is correct? From what has already been said there would seem to be no doubt that the accommodation is harmless in the production of the increase of tension in the eye that occurs during near work.

Foerster won the credit of demonstrating that we have a valuable prophylactic means for the treatment of myopia in the diminution of the convergence by the full correction of the refractive error by glasses, and most modern ophthalmologists have accepted his conclusions. *The full correction of the myopia is the first step in the treatment of the individual case.*

Some practical points of advice may be given. As long as the patients are young they should be instructed to wear the fully correcting glasses for both distance and near. Some of them will not be satisfied to do so at first. The increase of the visual acuity is distinctly inconvenient to those who have hitherto seen everything in a soft indistinctness. In addition to this they notice astigmatic distortions when they look obliquely through the ordinary concave lenses, but this sort of trouble may be avoided by the prescription of periscopic glasses. Such a patient must also learn to reestablish the correct relations between accommodation and convergence. A myope of 4 D reads without accommodation at 25 cm, at his far point, and has little or no tendency to converge, but with his fully correcting glasses he has to accommodate 4 D and to converge to 25 cm. As the result of all these things the myope may be unable to work at first with his full correction, so it is advisable to call his attention to this sudden and total change of the optical conditions, but these troubles usually pass away in a few days.

If we have to prescribe glasses for a myope who has reached an age in which the decrease of the range of accommodation is already perceptible, the presbyopic factor must naturally be taken into account for near work. A person with 4 D of myopia who is 60 years old and has no accommodation needs no glass to enable him to read at 25 cm.

Many myopes have a peculiar fear of strong glasses. During a careful test of the refraction they are very often heard to say "the glass is too strong." The preconceived idea of the harmful effect of strong glasses is still widespread even among physicians and ophthalmologists. This is a prejudice to be combated. We know that the accommodation does not increase the intraocular tension and that it is to the advantage of the myope to wear glasses that fully correct the refractive error, because it is only in this way that the best vision can be obtained. Still some persons who are very nearsighted manifest a great aversion to the full correction and cannot overcome this in spite of their efforts; in such cases, which are exceptional, we are obliged to prescribe weaker glasses for constant use, and especially for near work.

What therapeutic effects have been produced by the wearing of the full correction? The consistent application of this rule promises to produce the same effect as the measures that have been undertaken in school hygiene, to at least reduce somewhat the degree of the myopia. The hope has been cherished that we have perhaps in the full correction the means whereby to check the progress of myopia, but this hope has not been, and cannot be, perfectly fulfilled. Yet the full correction is one of the most important measures to be taken in the individual treatment. For example, *Pfalz* saw an average increase of the myopia of from 0.14 to 0.17 D in 3.3 years in eyes that were fully corrected for near work, and an increase of from 1.45 to 1.91 D in 4 years in eyes that were not fully corrected. Other statistics give similar results. The full correction is not treatment directed to the etiology of the disease, and therefore cannot prevent altogether the progress of myopia, but as it enables myopes to see distinctly at different distances in an instant, and above all to read and write at the greatest possible distance, it is a great aid.

Aside from the prescription of the correct glass there are various hygienic demands to be taken into consideration, most prominent of which is the selection of the life work, in the choice of which the physician assumes not a little responsibility. Less often the ophthalmologist is consulted concerning the advisability of marriage of myopes. Other considerations must decide this question, although rather more nearsighted children are born of nearsighted parents than of others. I also advise myopes, especially while they are young, to have their eyes reexamined every year to see if any change of glasses is needed.

The treatment of myopia cannot be said to be finished with such hygienic advice and the prescription of the proper glasses. On the contrary medical treatment often begins after pathological changes have appeared in the chorioid, retina and lens. But the advocacy of these two important points, maintenance of the measures of school hygiene and early full correction, is the performance of a great, one may say a national, duty.

197. The Operative Treatment of High Myopia

This patient tells us that both of his eyes were operated on some months ago on account of a high degree of myopia. Before the operations were performed he needed to wear very strong glasses for distance, but could see well close to his eyes without them; since the operations he has been able to see well at a distance with the naked eye, but needs glasses for near work. A few days ago the vision of one eye suddenly became very bad.

Hitherto we have been dealing only with the hygienic measures adopted in the schools to guard against myopia and with its treatment with glasses, but there is also an *operative treatment* to be considered.

Both eyes of our patient present the characteristic signs of aphakia; a deep anterior chamber, a tremulous iris, a deep black pupil. Traces of the lenticular capsule can be seen at the margins of the latter. Formerly he wore a very strong concave glass, he says it was number 20, but now he sees best at a distance with one eye without any glass. That eye is therefore emmetropic and it has a vision of 0.1. With the other eye he can see only movements of the hand, and by means of the ophthalmoscope we can see within it an extensive detachment of the retina. The changes characteristic of excessive myopia are to be seen in the head of the optic nerve and in the region of the macula of the other eye. The lenses have therefore been removed from both eyes of this rather young patient.

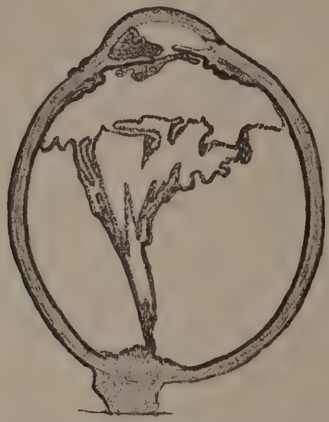

Fig. 175.—Detachment of the Retina Following Operation for High Myopia.

The operative treatment of high myopia consists of the removal of the transparent lens from the eye.

This operation is based on the experience of ophthalmologists for the last two hundred years, for even as long ago as that the important observation had been made that persons who were highly myopic and had to be operated on in advanced life for senile cataract did not need convex glasses in order to be able to see well at a distance, as did other cataract patients. The operation was performed in individual cases at that time, and more frequently in the fifties of the last century, as by *Weber* and *Mooren*, but its general extension has taken place more recently under the renewed impulse given to it by *Fukala* and *Vacher*. I no longer advise my patients to submit to this operation, but it is frequently performed by other surgeons.

The technique of the operation does not differ from that for the removal of the lens for other reasons, but each operative intervention on the highly myopic eye must be performed with special care and precaution. We may choose between two procedures: We may extract the lens forthwith, leaving a round pupil, by means of the flap operation, or through an incision made with a keratome, or we may first perform a discission of the capsule of the lens, so that the latter will begin to swell and grow opaque, and after this has proceeded far enough we can expel the swollen lenticular masses through an incision made with the keratome. Most surgeons prefer the latter operation. Injuries to the vitreous, as well as prolapse of the vitreous or of the iris, must be carefully avoided. The first thing to be decided therefore is the choice of the way in which the operation is to be performed.

The next question is: What change of refraction have we to expect after the removal of the lens from a myopic eye? We know that an emmetrope develops a hypermetropia of 10 or 11 D after loss of the lens, as after the operation for senile cataract, and that he needs a convex glass of this strength to enable the eye to see at a distance, as is proven by optical calculations, as well as by numberless clinical experiences. The conditions are somewhat different in myopia. The highly myopic eye apparently loses a larger part of its refractive power with the loss of its lens than the emmetropic eye. Thus it is a fact that myopic eyes which have needed glasses of from —8 to —20 D for distant vision become emmetropic after removal of the lens. It has been erroneously believed that this was because the index of refraction of the lens of a myopic eye was greater than that of an emmetropic eye, but it has been proved by a study of the optical conditions, as well as by the direct determination of the index of refraction of the lens, that the refractive power of the lens in the one eye is the same as that of the lens in the other. If this were not so we could not know approximately what optical result to expect after an operation for myopia. The change in the refraction after the removal of the lens from a myopic eye depends on fixed optical laws. Various tables have been given for the calculation of the degree of ametropia produced with different lengths of the axis of the eyeball, of which I present two.

Correcting Glass for the Aphakic Eye 12 mm from the Cornea	Axial Length	Correcting Glass for the Aphakic Eye 12 mm from the Cornea	Axial Length
+16 Dioptres	21.288	+5 Dioptres	27.671
+15 Dioptres	21.805	+4 Dioptres	28.326
+14 Dioptres	22.343	+3 Dioptres	28.994
+13 Dioptres	22.899	+2 Dioptres	29.689
+12 Dioptres	23.439	+1 Dioptres	30.380
+11 Dioptres	24.040	0 Dioptres	31.095
+10 Dioptres	24.617	—1 Dioptres	31.827
+9 Dioptres	25.179	—2 Dioptres	32.576
+8 Dioptres	25.787	—3 Dioptres	33.334
+7 Dioptres	26.406	—4 Dioptres	34.126
+6 Dioptres	27.015		

From this table it may be seen that if the axis of an eye is elongated 6 mm, say from 24 to 30 mm, an increase of the refraction is produced which is equivalent to a difference of 10 D in the glasses. An elongation of 1 mm of the axis of an eye that contains a lens corresponds to an increase of the refraction of 3 dioptres. When the normal eye containing a lens has become so myopic as to have an axial length of 30 mm its myopia amounts to 18 D. Hence the same change in axial length results in a change of refraction about 1.8 greater in an eye that contains a lens than in one that does not. The emmetropic eye needs a glass of +10 D for distance after the loss of its lens, the eye with 18 D of myopia just becomes emmetropic when its lens has been removed.

We may use the following table, calculated by *Hirschberg* for the reduced eye, to ascertain for practical purposes what refraction is to be expected after the removal of the lens.

Length of the Axis of the Eye	Refractive Condition of Eyes Containing Lenses	Refractive Condition of Aphakic Eyes
24	E	+10.0 Dioptres
25	M= 3 Dioptres	+8.5 Dioptres
26	M= 6 Dioptres	+7.0 Dioptres
27	M= 9 Dioptres	+5.5 Dioptres
28	M=12 Dioptres	+4.0 Dioptres
29	M=15 Dioptres	+2.5 Dioptres
30	M=18 Dioptres	+1.0 Dioptres
31	M=21 Dioptres	almost E
32	M=24 Dioptres	—1.5 Dioptres
33	M=27 Dioptres	—3.0 Dioptres
34	M=30 Dioptres	—4.5 Dioptres

We make the assumption—which is sufficiently accurate for practical purposes—that the change of refraction caused by a change of 1 mm in the length of the axis is about half as great in the eye that contains a lens as in one that does not. According to *Ostwald* we can also calculate the refraction of an axially myopic eye after removal of its lens by "dividing the number of dioptres of the glass needed to correct the myopic eye by 2 and then subtracting the quotient from 11 D, that is from the refraction of the emmetropic eye that has been made aphakic."

Some important conclusions as to the indications for the operation for myopia may be drawn from these tables.

Up to what degree of myopia is the operation permissible? Let us take an example. Suppose an operation to be suggested on a patient who has 9 D of myopia. After the removal of the lens he would have a hypermetropia of 5.5 D. Has anything been gained? Before the operation such a patient has good near vision without glasses and, if his accommodation is sufficient, has both distant and near vision with one and the same concave glass. After the operation he can see distinctly neither at a distance nor near by without glasses, and furthermore he needs two different pairs of glasses for distant and near vision, which he is continually obliged to change. Nothing more is needed to show that this operation is contraindicated in the low and medium grades of myopia by this purely optical reason. Although the views concerning the limits within which the operation is justifiable vary a great deal we can safely say that it should never be done for less than 14 D of myopia.

The second most important indication as to the advisability of the operation is furnished by the intraocular condition of the eye, especially by the condition of the retina. The rule of conduct is that the eye must be free from all opacities in the vitreous and that the fovea must be intact. It has been claimed, as I think erroneously, that retinal lesions may be favorably influenced by the removal of the lens. At all events the test of vision taken in conjunction with the ophthalmoscopic examination furnishes our best indication for or against the operation. The eye must be able to read easily the finest print. If this is not the case, if only the larger print can be deciphered, an operation is contraindicated, because the visual results that can be obtained will not come up to the expectations. Even when the visual acuity is impaired the larger letters can still be made out because the patient obtains an enlargement of the retinal images by drawing the book very close to the eye. After the lens has been removed this will be possible only by means of an extremely strong convex glass. Anyone who has seen the unfortunate results thus produced will be careful in his observance of the indications for the operation.

It seems plain to me, as it does to many other ophthalmologists, that the other eye should exhibit no serious visual trouble when the operation for myopia is to be performed. If the macula of the second eye is altered in any way so that its vision no longer corresponds to its objective condition I do not think that an operation should be advised.

Quite a number of patients have had both eyes operated on, and frequently the surgeon has been compelled by the patient himself to operate on the second eye. Conflicting views are held in regard to whether this is advisable. When only one eye has been operated on the patient can use that one to see at a distance and the other for near work. A good stereoscopic vision is certainly lacking in such a case, but this is not of very great importance to the person who is highly myopic. Personally I never advise an operation on both eyes. The desire of many myopes for the operation on the second eye can readily be understood when we take into account the advantages that beckon them after an operation has been successfully performed on the first.

The tables that have been given above concerning the change of refraction after extraction of the lens in myopia furnish us not only with the first indication, that only cases in which the myopia has reached a certain degree should be operated on, but also with the first advantage to be gained by the operation after it has been performed. When the person suffering from high myopia who has been able to see at a distance only with difficulty by the aid of the strongest glasses, has been dependent on his glasses to guide his footsteps, and has had to endure all of the other discomforts associated with the wearing of glasses, suddenly is able to see well at a distance without glasses, he has won a marvellous prize, the value of which cannot be appreciated by those to whom a normal eye is a matter of course.

The patient who has been operated on for myopia very often enjoys another advantage. He is not only able to see at a distance either without a glass or with a weak one, but he also obtains a delightful increase of his visual acuity. The reason of this is: When the lens is removed from a normal eye the anterior focal distance is increased, in the schematic eye, from 15.5 to 23.2 mm. The visual acuity is directly proportional to the anterior focal distance, the value for the minutest retinal image remaining the same. Hence the absolute visual acuity of the schematic eye that contains a lens is to that of the aphakic schematic eye as 15.5 : 23.2, i.e., as 1 : 1.15. The following table gives some calculations appertaining to this point.

The visual acuity of an eye which after removal of the lens is corrected by

$$\left.\begin{array}{r} +1=1.485\\ +3=1.455\\ +5=1.428\\ +7=1.402\\ +9=1.375\\ +11=1.351 \end{array}\right\}$$

of that found at the anterior focal point with the correcting glass before the removal of the lens.

Hence a highly myopic eye which has been rendered nearly emmetropic by the extraction of its lens may theoretically have a visual acuity 1.5 times greater than that which it possessed before the operation. It remains to be proved whether this increase of the visual acuity is to be explained by the enlargement of the size of the image alone. The view has been expressed that the working capacity of the retina itself may increase after the operation for myopia. According to *Schanz* the myopic eye that has been operated on should have, in comparison with one that has not been treated in this manner, a small pupil of egress and a large one of ingress, by means of which the dispersion is decreased and the light strength of the retinal images increased. It also happens that unexpected changes in the visual acuity appear in the test with concave glasses when the patient looks through the glass excentrically or obliquely. However this may be, whatever conditions may cooperate after the operation for myopia, the fact is that a great many patients who have been relieved of their nearsightedness in this manner exhibit a remarkable and delightful increase of their visual acuity. In view of these advantages it may seem surprising that I have not the courage to advise a person with high myopia to submit to this operation.

The least important disadvantage of the operation is the loss of accommodation. This is not to be taken seriously into account, for the region of accommodation is very small and lies close to the highly myopic eye, so that no use is made of it.

The question whether the removal of the lens is able to prevent an increase of the myopia is much more important. It has been claimed that a harmful factor is eliminated with the abolition of the accommodation, but as we know that the accommodation can produce no increase of tension within the eye this point cannot be urged in favor of the operation. The ciliary muscle remains unchanged after the removal of the lens and can contract in the aphakic eye during movements of convergence. The only point is that convergence no longer takes place after the lens has been removed, so that the compression of the eyeball by the extrinsic muscles is lessened. But the fundamental disease, the tendency of the posterior portion of the sclera to yield, remains unaffected. An increased stretching of the eye has been definitely found many times after the operation. For example, the refraction in *Scheffels'* ten cases increased from 0.5 to 2.0 dioptres in the course of from two to four years. The changes in the fundus have also been observed to undergo further development. For these reasons I do not believe that the removal of the lens is able to check the advance of the myopia.

All this might not be so bad, for a slight increase of the refraction could easily be corrected by glasses, but the main question, by the answer to which the operation for myopia must stand or fall, is whether the danger of detachment of the retina becomes greater or less. The greatest danger in excessive myopia is detachment of the retina. No equivalent results attend the operative removal of a lens from a highly myopic eye, be the technique never so faultless. Sometimes opacities appear in the vitreous of such eyes years later. Opinions are still very much divided as to whether this danger is increased or not by the operation. Many, especially *Vacher*, have claimed in an incomprehensible manner that the operation exerts a protective action against the occurrence of a detachment. It is generally acknowledged that this is not true. It is a fact familiar to every ophthalmologist that detachment of the retina takes place in many such eyes without operation. Cases have been known in which the operation had been decided on and the detachment took place before anything had been done to the eye. Most of the patients who have been operated on and later suffered from detachment have ascribed the latter more or less to the operation, and they have often been inclined to sue the surgeon. I have recently known damages to be awarded against a colleague. *It cannot be denied that the operation for myopia as such increases the danger of detachment of the retina.* That this question could not hitherto be answered with perfect clearness does not, in my opinion, speak favorably for the operation. Statistics show that the percentage of postoperative detachments of the retina is greater than that of spontaneous detachments in highly myopic eyes. The statistics of *Froelich* give the ratio as 3.3 : 1.25%. According to *Fischer* the percentage of spontaneous detachments in myopes that have not been operated on is 0.5%, in those that have been operated on 5.5% during the first year. According to my own notes also, detachment of the retina is more frequent after the operation than during the spontaneous course of high myopia. The disadvantages and the dangers of the operation for myopia outweigh the advantages, and the enthusiasm with which the operative treatment of high myopia was greeted in many places is beginning to notably abate. It does not lessen, it augments the danger of detachment of the retina. This is the reason I do not dare to advise this form of treatment. I believe it to be an unnecessary challenge to fate, which in many cases may leave contemptuously unnoticed the deeds of man to strike with ruthless hand when least expected.

198. Hypermetropia

I. The Course of the Rays of Light in the Hypermetropic Eye and the Measurement of Hypermetropia

This boy has one eye under the influence of atropin. Before the instillation of the drug he was able to see equally well at a distance with both eyes with the same glass.

The first fact that we learn is that he has perfect vision with the eye that is not under atropin. The second is that the same eye sees just as well with a convex glass of 2 D, which shows it to have a hypermetropia of 2 D. Hypermetropic refraction may a priori be due to: 1, a shortening of the axis of the eye; 2, a lessened curvature of the cornea; 3, a decreased curvature of the surface of the lens; 4, a low index of refraction of the lens and of the aqueous; 5, too high an index of refraction of the vitreous; 6, too great a distance from the cornea to the lens; 7, absence of the lens. We will consider here only the first form. Hypermetropia has already been defined as the condition of an eye in which the parallel rays of light that fall upon the cornea would come to a focus behind the retina when the accommodation is at rest. The far point lies behind the eye. Strictly speaking therefore this boy cannot focus parallel rays on his retina, he must see badly at a distance, for his eye is a priori adapted only to focus rays on the retina which fall upon the cornea with a certain degree of convergence. The far point of the hypermetropic eye is situated at the place where these rays, if prolonged, would meet behind the retina. The distance to this far point gives us the degree of hypermetropia, as it is a measure of the convergence of the rays to which the eye is adapted. The greater the hypermetropia the more convergent must be the rays that enter the eye in order to focus on the retina and the nearer to the posterior pole must the far point be situated.

As the far point of the hypermetropic eye lies behind the eye we designate it with the minus sign, —R. The rays must be directed toward, or, what is equally correct, must seem to come from, the far point.

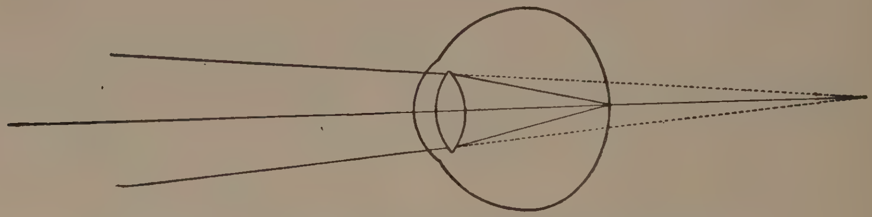

FIG. 176.—Course of the Rays of Light in Hypermetropia.

In spite of the faulty construction of his eye this boy is able to focus parallel rays of light upon his retina and to see well at a distance by increasing its refractive power by means of the action of his accommodation. Hence, *hypermetropia is the condition of the refraction in which the lens must assume a greater curvature when the eye is looking at objects in the distance.*

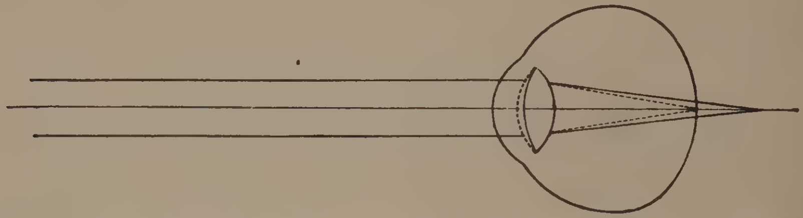

FIG. 177.—Self Correction of Hypermetropia by Means of the Accommodation.

The hypermetrope can relax this effort of the accommodation while looking at objects in the distance when convex glasses are placed before his eyes, because the glass placed in front of his eye takes the place of the supplementary lens secured otherwise by a bulging forward of the crystalline lens.

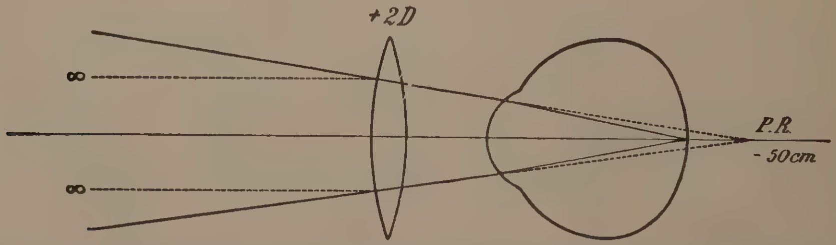

FIG. 178.—Substitution of a Convex Glass for the Accommodation in Hypermetropia.

This reveals one method by means of which we can measure the degree of the hypermetropia, for it is evident that we need for its correction a convex glass the focal distance of which coincides with the far point of the eye, or the focal distance of which is the same as the distance of the far point from the eye. *The convex glass which corrects the hypermetropia gives us* *through its focal distance the position of the far point and the degree of hypermetropia.*

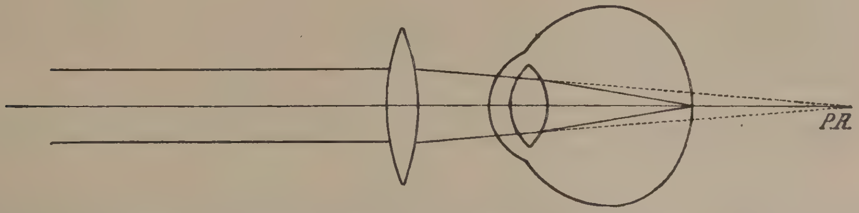

FIG. 179.—Correction of Hypermetropia with Glasses: Parallel Rays of Light Are Made to Converge toward *P. R.*

In this patient the convex glass necessary to correct the hypermetropia has a focal distance of 50 cm and the boy has therefore a hypermetropia of 2 D. We proceed in a manner exactly analogous to that followed in the measurement of myopia in that we place one after another stronger and stronger glasses before the eye; the only difference is that while we take the weakest concave glass as the measure of the error of refraction in myopia, we measure the hypermetropia by means of the strongest convex glass with which the best vision is obtained.

II. Manifest, Latent and Total Hypermetropia

When we turn to the eye that is under atropin we find that it needs 4 dioptres instead of 2 to enable it to see well at a distance. The hypermetropia has been made greater by the paralysis of the ciliary muscle induced by the atropin. This is a characteristic phenomenon of the youthful hypermetropic eye. Such a tonicity of the ciliary muscle develops in a hypermetrope that it cannot ordinarily be relaxed because he has been compelled from childhood to accommodate continually. We can cause a portion of the accommodation to relax by the interposition of convex glasses, but it is not able to yield entirely, a portion always remains active and conceals a part of the abnormal refractive condition of the eye. It is only when we actually paralyze the muscle with atropin that we learn the true degree of hypermetropia. We call the portion that is found by the ordinary test of vision with the strongest convex glass that is accepted the *manifest hypermetropia.* In this case the convex glass revealed only 2 dioptres, because the boy relaxed his accommodation only that amount. We designate as *latent hypermetropia* that portion of the tension of the accommodation which did not relax in the test with glasses. The true amount of the error of refraction, the *total hypermetropia,* is the sum of the manifest plus the latent. In this case we have ascertained the total hypermetropia by means of the action of atropin. It must be taken into account that when the pupil is so widely dilated the refraction is influenced by the flattening of the peripheral surfaces of the cornea and lens, so it is a better plan to determine the total hypermetropia either with the ophthalmoscope by the direct method, or by means of skiascopy, when the pupil is small.

These conditions are important in the treatment, for the ratio between manifest and latent hypermetropia is variable. It may be changed, for example, by the wearing of glasses. When stronger and stronger glasses need to be prescribed for a hypermetrope it is because the latent hypermetropia is gradually growing less and the total may finally become manifest. The manifest portion of the hypermetropia depends mainly on the amplitude of the accommodation and the age. Up to the age of 25 years half of the total hypermetropia may be said to be manifest, but the older the patient grows the greater will the manifest portion become, and after the forty-sixth year the manifest is the same as the total in most cases.

III. The Selection of Glasses and the Troubles of Hypermetropia

The knowledge of this ratio is important because the question naturally arises what glass to prescribe. We should give the full correction in the treatment of myopia, but this is not, to say the least, judicious in many cases of hypermetropia. It is possible by a gradual increase of the strength of the glasses to be able to correct the entire hypermetropia and enable the patient to see at a distance without the use of his accommodation, but if it should ever happen to be necessary for such a hypermetrope to see at a distance without glasses experience has proved that this tax upon his accommodation is much more severe than it is when he has been accustomed to make his ciliary muscle work rather more by wearing glasses that were not perfectly corrective. For this reason it is better to prescribe for hypermetropes who are less than 25 years old only the glasses that correct the manifest portion of their errors of refraction. The total hypermetropia should not be corrected until after that age.

There is another reason why we have so much to do with hypermetropia. The range of accommodation is just as great as it is in emmetropia or myopia, because this depends simply on the ability of the lens to assume a globular form, but as the hypermetrope is obliged constantly to use a portion of his accommodation for distance it is evident that *presbyopia must begin earlier in hypermetropes than in emmetropes.* Let us take an example: At the age of 33 the emmetrope still has a range of accommodation of 6 D at his disposal, P is at the distance of 17 cm. A hypermetrope of 2 D at the same age, who has the same amplitude of accommodation, needs two of the six dioptres for distant vision and his near point is therefore 25 cm from his eye. Thus it is that after the age of 45 we are frequently obliged to prescribe for hypermetropes different glasses for distant and near vision. Under certain conditions we must deviate from the rule to correct only the manifest portion of the hypermetropia in young people. A stronger glass may be necessary in feeble and anæmic persons. We must also give the full correction if possible when there is a pathological tendency of the eyes to convergence, in strabismus convergens, because this tendency may be obviated by doing so. In the high degrees of hypermetropia it is also better in most cases to correct a larger part of the refractive error than is manifest.

Ordinarily the hypermetropia is of a low or medium grade, it rarely amounts to more than 6 or 7 dioptres. When the eye is smaller than this we usually find in it other congenital anomalies, such as coloboma or microphthalmos, but cases are met with in which the hypermetropia is from 8 to 20 dioptres in eyes that are otherwise healthy. Such persons usually behave in reading as though they were nearsighted, they hold their books very close to their eyes and have in consequence been thought to be highly myopic in many cases. *Salzmann* has shown that this peculiar vision of hypermetropes is due to the fact that the fault of focus increases in a much less degree than the size of the retinal images.

We must proceed carefully in the fitting of glasses. Here is a student who complains that his near vision is not distinct for any great length of time. He can read for a while, then the letters blur before his eyes, he pauses in his work, begins to read again, the letters blur again after a shorter interval, so that he is obliged to lay aside his work more and more often and for longer intervals. In addition to this he has an unpleasant sensation of pressure in his eyes, and even pain, which radiates into the brow. We find that he has hypermetropia. This is the clinical picture of *accommodative asthenopia.* It is caused by the overstraining of the ciliary muscle, and the blurring of objects in prolonged near work is due to the fact that the overstrained accommodation gives way and the eye then ceases to be correctly focussed. This weariness of the ciliary muscle is explained by the great demand made upon it by the hypermetrope. There is no fixed limit at which asthenopic troubles appear, sometimes they are present when the degree of error is very low. Other factors take part such as the general condition and the nature of the employment, and it has already been pointed out that the other muscular conditions of the eye, such as the connection between the convergence and the accommodation, must be taken into account. We are often obliged to prescribe stronger convex glasses than are accepted in cases of accommodative asthenopia in order to aid the eye.

IV. Apparent Optic Neuritis in Hypermetropia

When the hypermetropic eye is carefully examined it may be easily recognized that it is rather smaller in all diameters than the normal, particularly in the higher grades. The principal cause of hypermetropia is a shortening of the axis of the eye. The anterior chamber is shallower in the hypermetropic than in the normal eye, because the lens, which develops independently of the form of the capsule of the globe, is large in comparison with the small eye. I need only recall what was said in regard to this matter under the subject of glaucoma. In addition to this the ciliary muscle is constantly in a certain state of contraction in hypermetropes, and consequently the lens swells forward rather more and so contributes to the shallowness of the anterior chamber. Above all we find with the ophthalmoscope in a number of cases of hypermetropia a change in the papilla which has been termed pseudoneuritis, and has a certain diagnostic importance because of its resemblance to optic neuritis. The papilla appears rather red and swollen with hazy margins. It may be differentiated from true neuritis by the fact that it remains stationary, that it does not result in atrophy, that no white spots or hemorrhages are to be found associated with it, and that it causes no functional disturbance.

199. Astigmatism and Its Determination

Much time and labor is saved in the functional test of the eye by first ascertaining the refraction objectively with the skiascope. Our first patient presents a condition to which we have as yet paid no attention. The skiascope informs us that the refraction of the horizontal meridian of his eye is quite different from that of the vertical. We would strive in vain to improve his vision by means of spherical glasses, for the position in which such an eye finds itself relative to its vision may be made clear by this plate, which shows the course of the rays of light in astigmatism.

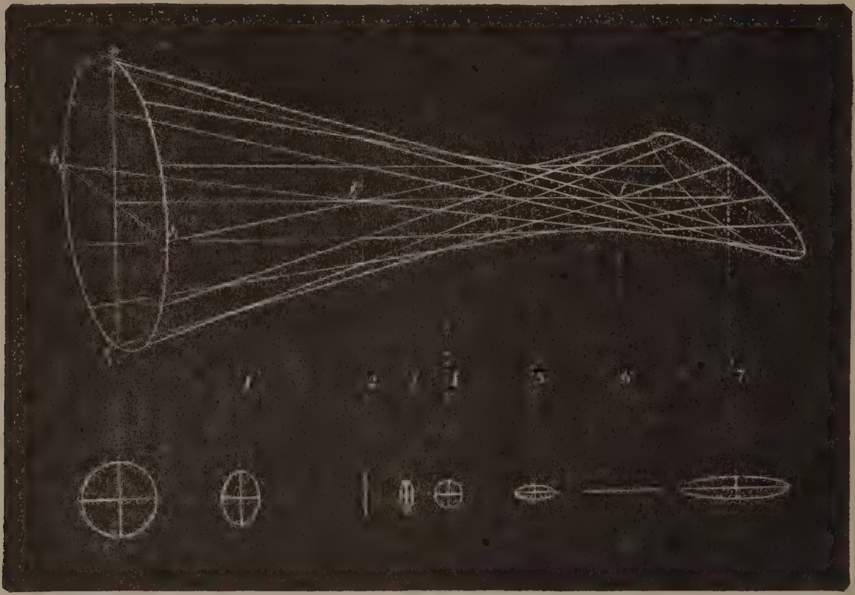

FIG. 180.—Course of the Rays of Light in Astigmatism.

The simplest and commonest form of astigmatism is caused by a difference in the curvature of the two principal meridians of the cornea which are perpendicular to each other.

For example: The rays that pass through the vertical meridian of the cornea focus at *f*, those that pass through the horizontal meridian at *f′*, in consequence of the greater curvature, hence the eye is unable to unite into one common focus at any point the parallel rays that fall upon it. The two meridians in which the refraction is greatest and least are called the two principal meridians. The meridian next adjoining the vertical has a somewhat greater curvature and the curvature of each meridian increases up to the horizontal. The image cast by a cornea that is curved in this manner can be sharp nowhere. The form of the image depends on the situation occupied by the retina in this system. If, for example, it were situated at the place marked 1 the transverse section of the cone of rays would form a vertical ellipse. At *f*, the anterior linear focus, the image of a point would form a vertical line, and so on. The interval between *f* and *f′*, the anterior and posterior linear foci, is called the interfocal space.

It is evident that under such conditions the vision must be considerably impaired in astigmatism. According to *Muegge* the relations between astigmatism and impairment of vision are as follows:

Astigmatism	Vision	Astigmatism	Vision
Less than 1 D	0.8	2—3 D	0.4
1—1.25 D	0.6	>3 D	0.3
1.25—1.75 D	0.5		

In most cases the cause of astigmatism is an abnormal curvature of the cornea. *Most commonly the vertical meridian has a greater curvature than the horizontal.* This anomaly is known as *direct astigmatism.*[1] More rarely the horizontal meridian has the greater curvature and the condition is then called *inverse astigmatism.*[2] But the cause may also be situated in the lens, and this must be borne in mind when we find a difference between the corneal and the total astigmatism.

Whatever the cause may be, the vision of such an eye is considerably impaired. The objects seen may appear distorted, or simply indistinct, according to the position of the bundle of rays that the patient uses. *Hess* has proved that the image of a letter may be perceived more easily from the focal circle than from the linear focus. It can be established in many cases that heredity plays a great part in astigmatism. Even the direction of the principal meridians may be hereditary. Two practical points interest us: 1, How to determine the astigmatism; 2, how to proceed in fitting glasses for its correction.

I. Determination of Astigmatism

We have to differentiate between *regular* and *irregular astigmatism.* In regular astigmatism there is a different curvature of the individual meridians of the refractive media, but the curvature in each meridian is uniform. In irregular astigmatism the curvature is not everywhere the same in one and the same meridian. We find irregular astigmatism chiefly in connection with corneal opacities, and will not take it into consideration at the present time.

To ascertain whether any considerable degree of astigmatism is present in an eye we first examine the cornea with *Placido's* disc, the concentric circles of which will appear to be elliptic in the reflection from the surface of the cornea if there is much astigmatism. The short axis of the ellipse indicates the principal meridian of the greatest curvature.

The presence of astigmatism may also be detected with the ophthalmoscope from the following signs. As the enlargement must vary when the curvature of the meridians of the cornea are of different strengths the papilla appears to be not round, but oval. In the upright image the papilla appears to be larger in the more strongly refracting meridian than in the one in which the refraction is weaker, so if the vertical meridian has the stronger curvature the papilla appears to be vertically oval. In the inverted image, on the contrary, the enlargement is greater in the less strongly refracting meridian, provided that the loupe is held nearer the eye than its focal distance, so that the papilla appears to be horizontally oval. We can determine the refraction of the individual meridians in the same way that we can estimate the total refraction in the upright image, by focussing upon the minute vessels on the papilla, but this method of measuring the astigmatism is quite difficult, the work can be accomplished much more easily with the skiascope.

The mirror is first rotated on its vertical axis until the refraction in the horizontal principal meridian has been learned, then it is rotated on its horizontal axis and the refraction in the vertical meridian determined. The following possibilities may be present.

There may be emmetropia in one principal meridian, hypermetropia in the other, the case then is one of *simple hypermetropic astigmatism.* If one meridian is emmetropic and the other myopic we have *simple myopic astigmatism.* Both meridians may be hypermetropic or myopic to different degrees, and then we have a *compound hypermetropic or myopic astigmatism.* If one meridian is hypermetropic and the other myopic the case is one of *mixed astigmatism.*

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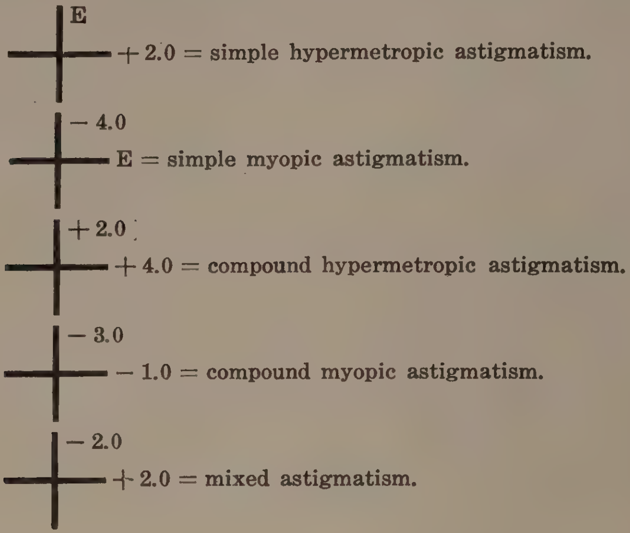
[1] [Or astigmatism with the rule—*F.*]

[2] [Or astigmatism against the rule—*F.*]

We indicate the condition found by the skiascope in the following way:


E
+ 2.0 = simple hypermetropic astigmatism.

− 4.0
E = simple myopic astigmatism.

+ 2.0
+ 4.0 = compound hypermetropic astigmatism.

− 3.0
− 1.0 = compound myopic astigmatism.

− 2.0
+ 2.0 = mixed astigmatism.

In this way we are informed at once about what glass the eye in question will need, but in many cases it is necessary to control this skiascopic finding and to determine the astigmatism rather more accurately. It is an advantage of the skiascopic method that we are able to learn at once the total astigmatism which is decisive in regard to the function of the eye and the fitting of glasses, but it is often difficult to ascertain it positively in this manner because the pupil may contract and the patient accommodate while he is looking straight ahead, and if the patient should look a little to one side, so that we get the refraction of the region of the papilla, we have not the conditions that correspond exactly with the refraction in the line of vision. There is still another important factor: Frequently the principal meridians are not exactly vertical and horizontal, but oblique. The expert notices this at once when he uses the skiascope, the apparent move-

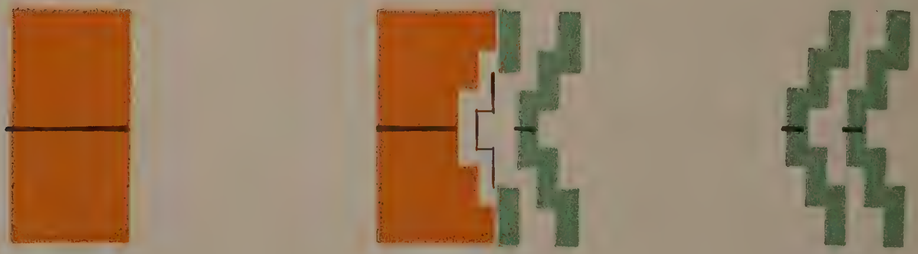


FIG. 181 a. FIG. 181 b. FIG. 181 c.

Determination of Astigmatism.

ment of the illuminated field is not in the same plane as the rotation of the mirror.

In such cases we can determine exactly the deviation of the principal meridians from their normal position by means of the *ophthalmometer* which was devised by *Helmholtz* and adapted to practical use by *Javal* and *Schioetz*. We measure with this instrument the images reflected from the cornea and find from them the curvature of the latter. Given a fixed size of the object and a fixed distance of the object from the cornea, the size of the reflected image changes only with the degree of curvature of the cornea. This curvature may therefore be calculated if the size of the reflected image is known. The necessary apparatus for this purpose is contained in the telescope which passes through the centre of the arc. This shows us the reflected images enlarged and doubled by means of a double prism. The objects may be made to appear red and green by means of red and green glasses.

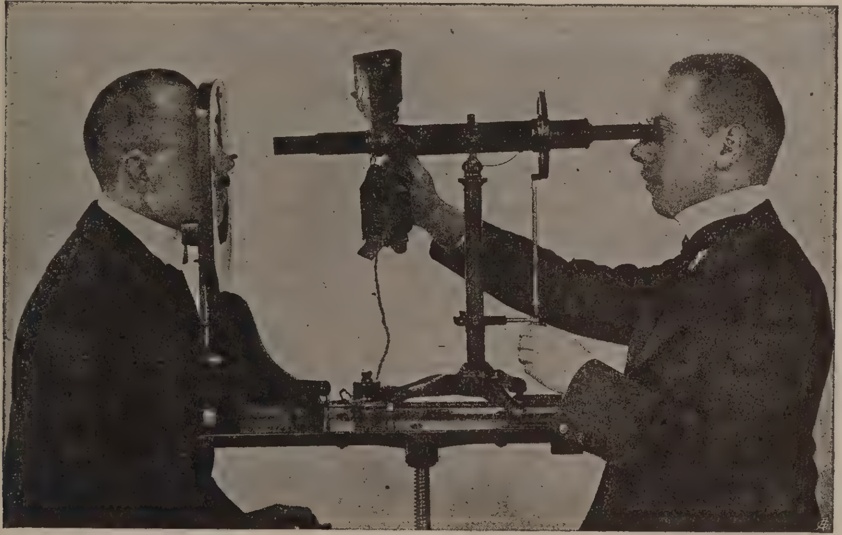


FIG. 182.—Measurement of Astigmatism with *Javal's* Ophthalmometer.

The patient places his chin on the chin rest and is directed to look with the eye that is being examined directly into the tube, which we point at the eye. We then move the apparatus near enough to the eye to be able to see the mires distinctly as we look through the tube. This is the primary position. We move the mires back and forth until we see that the images just touch. Then comes the second step. We must ascertain whether the principal meridians are oblique or not. This is done simply by noticing whether the bases of the mires are of equal height when the arc is in a horizontal position. If the bases of the mires do not coincide when the arc is horizontal the principal meridians are oblique. All that is necessary then is to turn the arc slowly until the bases of the two mires exactly coincide. We find in this way the direction of the principal meridians very accurately and can read the degree of obliquity from a scale that is attached to the instrument. It was decided at the last International Ophthalmological Congress that the notations of the meridians of each eye shall begin with 0 at the nasal end of the horizontal meridian and increase to 180° at its temporal end. After the direction of the principal meridians has been ascertained we need only to rotate the arc 90° and see whether the mires continue to touch. If the curvature of the meridian of the cornea now being examined is different the mires may overlap and one or more steps may be covered. The comparative size of the mire and of the steps is such that each step corresponds to one dioptre of difference of refraction. We therefore find by means of this instrument the position of the principal meridians, or their deviation from the normal, and the difference in their refractive powers expressed in dioptres, i.e., the astigmatism. On another scale we can read the length of the radius of curvature. The technique of using this excellent instrument can be learned in a few minutes. A slight difference of curvature between the horizontal and vertical meridians is present in the perfectly normal eye. In most cases the vertical meridian is more refractive than the other by 0.5 to 0.75 D. This amount of astigmatism is therefore physiological.

II. The Prescription of Glasses for Astigmatism

We first learn definitely in how far the astigmatism impairs the vision and what glass is best fitted for its correction from the functional test of such an eye.

It is plain that we cannot correct this error with spherical glasses, we need to use instead cylindricals. Imagine a segment cut from a cylinder; this forms a convex cylindrical lens. The rays of light that fall upon it in the plane of its axis undergo no refraction, while those that fall upon it in the plane perpendicular to its axis undergo the maximum of the refraction that corresponds to the curvature of the cylindrical surface. The same is true of the concave cylindrical lens, which represents a cast of the convex cylinder.

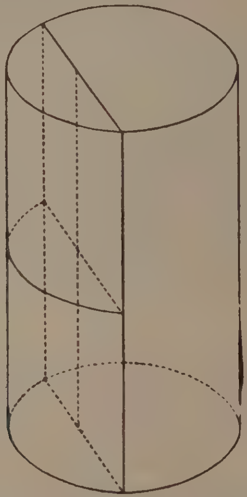
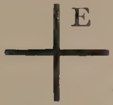


FIG. 183.—Cylindrical Glasses.

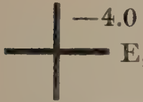
We can easily determine whether we are dealing with a convex or a concave cylindrical glass by the method of parallactic displacement. When we move it before our eyes in the direction of its axis there is no apparent movement of the object looked at through it, but when we move it in a direction vertical to its axis the apparent movement of the object is immediately perceptible. In order to facilitate the prescription of cylindrical glasses for astigmatism we take into account only the two principal meridians and pay no attention to the rest of the cornea.

Examples. When we find



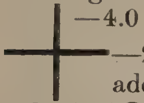
+2.0, that the eye is hypermetropic 2 D in its horizontal meridian and emmetropic in its vertical, it is plain that we must place before the eye a convex cylindrical lens with its axis vertical. Then the error of refraction in its horizontal meridian will be corrected, while the refraction in the vertical meridian remains unchanged.

In simple myopic astigmatism, e.g.,



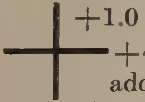
E, we prescribe a concave cylindrical lens with its axis horizontal.

In compound astigmatism we need a combination of spherical and cylindrical glasses. If we find with the skiascope



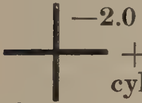
—2.0, there is a myopia of 2 D in both meridians together with an additional 2 D of myopia in the vertical meridian, so we prescribe —2.0 spherical ○ —2.0 cylindrical axis horizontal.

The conditions correspond in compound hypermetropic astigmatism, e.g.,



+4.0. There is a hypermetropia of 1 D in both meridians with an additional 3 D in the horizontal, so we prescribe +1.0 sph. ○ +3.0 cyl. axis vertical.

In a case of mixed astigmatism, e.g.,



+1.0, we may prescribe a priori +1.0 cyl. axis vertical ○ —2.0 cyl. axis horizontal, but can combine these figures and write +1.0 sph. ○ —3.0 cyl. axis horizontal.

The more experience we have the more certain we become of the fact that the astigmatic eye by no means always accepts the glass as best which is indicated by the skiascopic and ophthalmoscopic measurements. Very often the glasses chosen objectively and subjectively are markedly different. Various factors enter into the explanation of these differences between the ophthalmometrically determined and the subjectively ascertained astigmatism. Deviations of both surfaces of the lens from the spherical form play an important part. *Gullstrand* has shown that the influence exerted by the size of the pupil upon the refraction of light in the cornea does not suffice to explain these differences. The claim has frequently been made that the eye can compensate for a portion of the corneal astigmatism by a partial contraction of the ciliary muscle, but it has not yet been proved that a partial contraction of the ciliary muscle can take place. *Hess* has revealed sufficiently the sources of error that may be present in attempts to prove that it does. At all events we learn by experience that the patients themselves are best able to determine which glass is best fitted to them. *It is equally wrong to fit cylindrical glasses while the pupil is dilated by atropin.* In consequence of the dilatation of the pupil and the participation of the peripheral portions of the cornea and lens, glasses can then be fitted which cannot be borne by the eye under normal conditions. As a general rule we prescribe the weakest cylindrical glass with which the eye obtains its best vision, and I wish to call attention to the fact that many astigmatic eyes cannot obtain anything like normal vision even with the very best correction with glasses.

Finally we have a patient who has been operated on for cataract and needs to be fitted with glasses. Hitherto we have been contented to say that an eye that was formerly emmetropic develops after the removal of its lens an average hypermetropia of 10 D, and accordingly needs a +10.0 D glass to see clearly at a distance, but there is in the majority of cases in addition to the hypermetropia an astigmatism produced by the wound. The larger the section is made, and the closer it lies to the central part of the cornea, the greater is the resultant astigmatism. In the usual extraction upward the horizontal meridian of the cornea acquires a greater curvature, the vertical meridian a less. As the adhesion of the margins of the wound grows firmer and the region of the incision becomes flattened this astigmatism is accustomed to grow less as the weeks pass by. It is therefore the rule that the final glass should not be prescribed for a cataract patient until six or eight weeks have elapsed since the operation. As this astigmatism is inverse, or against the rule, we add to the necessary convex glass a convex cylindrical lens with its axis horizontal. Here is a point to note. Cataract patients often do not see as well with the spherocylindrical glasses made by the optician, as with the same combination of test lenses, although the prescription has been filled correctly. The optician grinds the double curvature of our biconvex lens upon the anterior surface of his glass and that of the cylindrical lens upon its posterior surface. According to *Dimmer* the spherical component of such a glass may readily be made too strong and the cylindrical too weak. According to *Ostwald* this clinical phenomenon is due to the fact that the correcting cylindrical glass for the aphakic eye is influenced a great deal in its effect by its relative position to the eye. At all events it is a practical point that we should test our patients who have aphakia and astigmatism with plano convex spherical glasses and that when we add the cylindrical glass we should place it next to the eye. Many attempts have been made to obviate astigmatism by means of an operation, but these have not given satisfactory results, so we have to content ourselves with the correction by glasses. In some cases in which we can obtain no useful vision in this manner, we can make use of stenopæic slits, as in keratoconus, by means of which an astigmatic eye often can obtain better vision.

A few words concerning irregular astigmatism, which we meet with in great variety, especially in connection with opacities of the cornea. In commencing opacity of the lens also the refractive powers of the different sectors of that organ may differ so much that the retinal images cast by them may be broken apart and may finally be perceived to be separate. Irregular astigmatism causes objects to appear to be distorted. It cannot be corrected with glasses.

200. The Test of the Peripheral Vision and the Disturbances of the Visual Field

All of the methods we have thus far employed, the test of the visual acuity, the measurement of the refraction, the test of the accommodation, and the determination of the amplitude of accommodation, deal primarily with the central vision. If we hold up a hand to one side of the eye, while the latter is looking straight ahead, we can perceive its movements with the periphery of the eye. This perception depends on the functional power of the peripheral parts of the retina, and the investigation of the peripheral vision is a necessary part of the complete functional test of the eye. The projection of the entire region of the retina that is sensitive to light we call the *field of vision*, which includes therefore that part of space from which the eye without moving can receive visual impressions. We have to test the visual field in different ways according to the capacity for vision that the eye possesses, which may be illustrated on these three patients, one of whom has a mature senile cataract, one an extensive detachment of the retina, and one commencing atrophy of the optic nerve due to tabes.

I. Test of the Projection in Mature Cataract

When a cataract has become sufficiently mature to render an ophthalmoscopic examination of the eye impossible we have to ascertain whether the retina still possesses normal functional power by means of the test of the projection. In spite of the opacity of the lens such an eye must be able to perceive the light of a candle in a dark room at the distance of several metres, and the patient must above all be able to tell us from what direction the light enters his eye. We throw light into the eye of this patient by means of a mirror and he perceives and states accurately the direction from which the light comes. It has already been stated that such a result as this is preconditional to success in an operation for cataract.

II. Control Test with the Examiner's Own Eye

Our second patient has an almost total detachment of the retina and can perceive nothing with the eye except movements of the hand. If we wish to learn the extent of the field of vision that has been preserved in this and similar cases we may proceed in the following manner: We cover the good eye and have the patient look with the bad one at our own; then we bring a large object, like a wad of cotton, with twitching movements from the periphery toward the centre in various directions, and we are thus able to learn from our own field of vision what part of that of our patient has been preserved.

III. Test with the Perimeter

If the eye still possesses central vision and a fair degree of visual power we test the field by means of the perimeter, an instrument of which several models are in use.

a. The Normal Field of Vision

The limits for the field of vision are fixed by the points situated farthest from the visual line from which the rays of light can excite a sensation of light within the eye. Under normal conditions the size of the field of vision is influenced by the following factors: 1, By the position of the lids; the field is enlarged somewhat when the upper lid is strongly raised or when we draw back the outer commissure; 2, by the position of the upper and lower margins of the orbit, the nose and the eyebrows; the visual field of a deeply sunken eye is somewhat contracted; 3, by the size of the pupil, which is of less consequence, except that when the pupil is very small the amount of light that enters the eye may be limited, and so the function of peripheral vision may be compromised; 4, the limits of the visual field may be enlarged a little during accommodation by the bulging forward of the anterior surface of the lens, as then rays can enter the eye which are intercepted by the iris when the eye is at rest; 5, by the form of the eye itself; the field of vision is sometimes smaller in axial myopia than in hypermetropia. But all of these conditions can scarcely be said to play any part when the question is to discover pathological conditions, for, as a rule, these are disclosed with sufficient clearness.

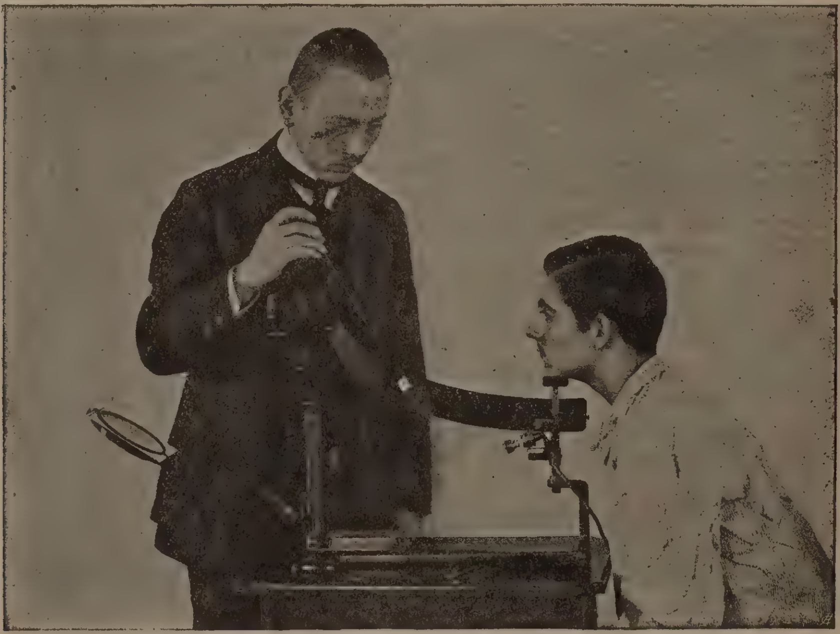


FIG. 184.—Perimetry

We begin by ascertaining the limits of the visual field for white and then for colors. The average extent of the field of vision may be said to be:

For white to the nasal side and above 60°, below 70°, to the temporal side 90°.

For blue to the nasal side and above 50°, below 50°, to the temporal side 70°.

For red to the nasal side and above 40°, below 40°, to the temporal side 50°.

For green to the nasal side and above 30°, below 30°, to the temporal side 40°.

b. Perimetry in Disease and the Most Important Disturbances of the Field of Vision

A defect in the visual field is called a *scotoma.* We distinguish first between a *negative* and a *positive scotoma.* We discover the negative by perimetry, the patient himself notices the positive as a dark shadow. This distinction is of importance in so far as we thereby obtain data in regard to the site of the disease. A positive scotoma, of which the patient complains, indicates that the lesion is situated in the eyeball itself. The scintillating scotoma is the only one that is positive and is due to a lesion in the brain. Negative scotomata are usually caused by lesions in the optic paths of conduction.

We also distinguish between an *absolute* and a *relative scotoma* for black and white and for colors. A scotoma is absolute when the patient is unable to see a black and white object in the portion of the field affected; it is relative when black and white objects can be seen but only indistinctly, and colors appear to be gray. The principal types of curtailment of the field of vision are: 1, Concentric contraction; 2, central scotoma; 3, annular scotoma; 4, sectorlike scotoma; 5, peripheral scotoma. The following points are diagnostic: We meet with concentric contraction in simple atrophy of the optic nerve, in the atrophic stage of choked disc, and to a high degree in pigment degeneration of the retina. It causes troubles of orientation in all cases of this nature, but such trouble is absent in the concentric contraction due to traumatic neurosis and hysteria. The troubles are only slight at the commencement of a concentric contraction of the visual field due to organic causes. In a case of central scotoma, in which the peripheral portion of the field is well preserved, we must try to find an explanation of the impairment of central vision in the ophthalmoscopic condition. The proof of the existence of a central scotoma is furnished by the fact that an object is seen better excentrically than in the visual line. When we place a small colored object before a patient in his visual line it is best to cover it at first, then to uncover it suddenly and ask him to name the color. Such a scotoma may be present either in one or both eyes. Unilateral scotomata are due in many cases to central diseases of the retina and chorioid, and attention is called to them, as a rule, by such symptoms as metamorphopsia and micropsia. The most important causes are central retinitis, central chorioiditis, and hemorrhage in the macula. Still a unilateral central scotoma may be due to a retrobulbar cause, such as multiple sclerosis, syphilis of the base of the brain, or the so called rheumatic affections of the optic nerve. Bilateral central scotoma usually indicates a disease of the papillomacular bundle, as in alcohol and tobacco amblyopia, in diabetes, and in the cachexia of carcinoma, but central affections of both retinæ may also be the cause. In annular scotoma the centre and the periphery are not affected. This form is met with chiefly in pigment degeneration of the retina, in the chorioiditis of hereditary syphilis, and in diseases of the posterior ciliary vessels. Sector shaped scotomata are mainly due to occlusions of certain vessels, and the limits for both white and colors usually coincide with the line of demarcation of the scotoma. Irregular sectors may also be met with in tabes and multiple sclerosis, but in these the limits for white and for colors are not apt to lie close together. Finally, the field of vision is contracted irregularly from the periphery in glaucoma and detachment of the retina. In glaucoma it usually begins on the nasal side. The conditions presented by hemianopic defects of the visual field have been described in another place.

201. The Test of the Light Sense

The phenomenon of the adaptation of the eye to reduced light is well known to everybody. When we pass from a brilliantly lighted room to one that has much less light, or is dark, we can see little or nothing at first, but gradually objects become more distinct. This phenomenon is due to the fact that the visual purple regenerates itself in the retina when the light is decreased, and as it augments in the outer segments of the rods of the retina the eye becomes sensitive to minute quantities of light. If this adaptation of the eye is studied more carefully we find that the sensitiveness of the eye in a dark room increases at first rapidly, then more slowly, and that it reaches its maximum in about twenty minutes. The physiological fact is also worthy of notice that the fovea has a slow power of adaptation. Thus it happens that after we have remained in the dark for some time we can perceive excentrically faint traces of light which disappear as soon as we look directly at them.

Serious disturbances in the adaptation of the eye to reduced light are met with in various conditions. They ordinarily take the form of a delay in adaptation, and it is this form with which we have most to deal. An increase of sensitiveness to minute quantities of light occurs only in persons who are totally color blind.

The usual disturbance is revealed clinically by the so-called hemeralopia, in which the patients see very badly as evening approaches, have faulty orientation, and sometimes are absolutely helpless. We find hemeralopia in pigment degeneration of the retina, in siderosis bulbi, and in the disease of the retina and chorioid due to hereditary syphilis. We must differentiate this kind of hemeralopia, which is due to a disease in the fundus, chiefly of the

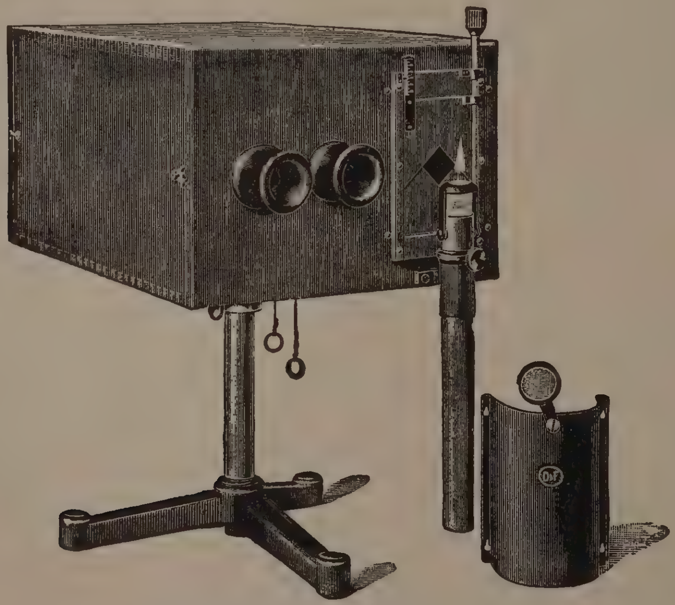


FIG. 185.—Foerster's Photometer.

retina, from those troubles that are caused by opacities of the cornea. Large, diffuse corneal opacities sometimes excite hemeralopic troubles when the pupils dilate in the evening and the peripheral portions of the cornea gain importance in the act of vision. On the other hand patients with purely central opacities of the cornea or lens may be able to see better in the evening, when the pupil is dilated (myctalopia). There is also a hemeralopia caused by a general nutritive disturbance. I refer to that described under xerosis of the conjunctiva. In this also the adaptation of the eye is delayed.

This boy, 16 years old, has been brought to us because he has exhibited symptoms of hemeralopia for several weeks. As soon as twilight comes on he is obliged to feel his way along in order to go home. The ophthalmoscopic condition is perfectly normal, but the boy is very badly nourished. When we wish to investigate in such a case as this whether a disturbance of the light sense is present we can simply darken the room and ascertain by our ordinary test types whether the visual acuity of the patient has decreased more rapidly than our own, which we know to be normal.

The conditions of adaptation may be tested more accurately by means of *Foerster's* photometer, which consists of a box with an opening in one of its narrow walls to which each eye is to be applied. Near one opening is a window 5 cm square covered with white paper. This paper receives the light from a candle enclosed in a lantern attached to the outer wall of the box. We can regulate the amount of light that falls upon the paper by means of *Aubert's* movable diaphragm, the opening of which can be reduced from 1500 square millimetres to 1 square millimetre. We can read the side length of the opening in the diaphragm on the scale that is attached. The object to be looked at, white stripes on a black background, is on the inner surface of the opposite side of the box. In order to enable the eyes that are not yet accustomed to the dark to see the object, the diaphragm is opened wide. As soon as the patient sees the object, the opening in the diaphragm is made smaller, the time and the width of the aperture noted. As the patient continues to look into the box the object gradually reappears and the opening in the diaphragm is again made smaller until the object has again disappeared. The effect of adaptation in such a hemeralope can be given in millimetres on the diaphragm in ten minutes. The light sense is so impaired in this patient that the opening in the diaphragm can be contracted only to 10 mm, although we can see the object when the aperture is smaller.

202. The Test of the Color Sense

It was shown in the examination with the perimeter that the outermost periphery of the retina is color blind. If in perimetry we move the color test object from the periphery of the field toward its centre we first notice only the movement of the object, and then gradually we become able to recognize the color. Under normal conditions the limits within which colors can be seen differ for each individual color. The field of vision is small for green, rather larger for red, still larger for yellow and largest for blue.

We have seen repeatedly that disturbances of the color sense may be acquired in diseases of the retina, and of the optic tracts, as in simple atrophy of the optic nerve. At first the field for white may be quite normal, although there is a distinct contraction of those for green and red. Hence the color test, especially the test for red and green, is frequently the first of all to disclose a pathological process.

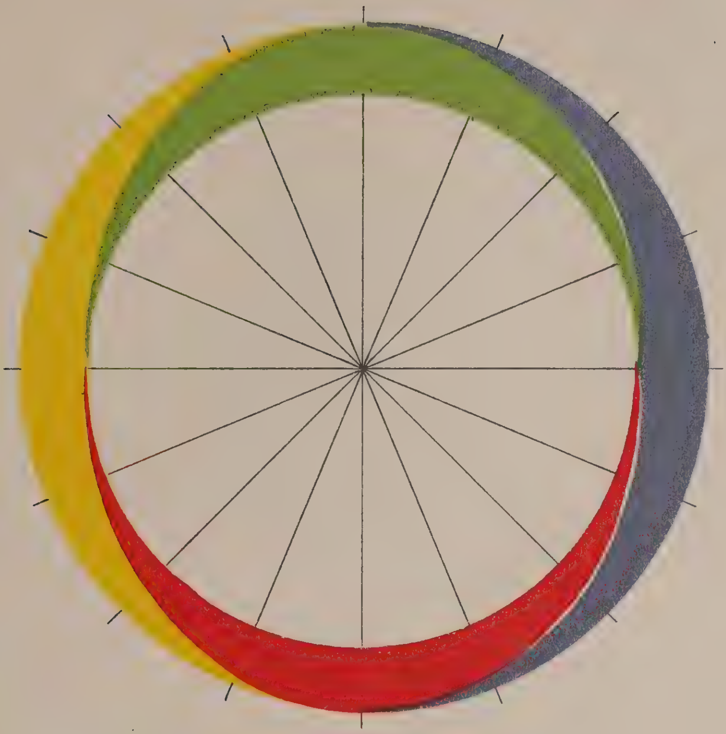
Congenital disturbances of the color sense are met with. The most common is the red green blindness. We have to deal with these anomalies when we have to determine whether employees of the railway, or persons connected with the army, have normal color sense.

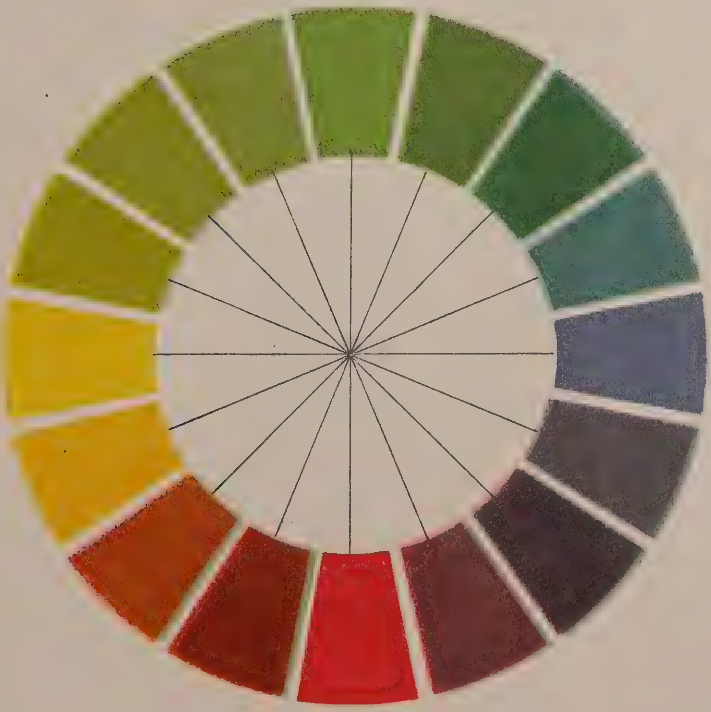
We can distinguish in every color its tone, its saturation and its brightness. By the term tone we refer to the quality of the coloring components, while the saturation is dependent on the ratio in which the colored and colorless components take part in the composition of the sensation of color. Thus we see in orange some red together with the yellow, and in another yellow some green.

There are only four colors which we may designate, according to *Hering*, as basal or primary, the appearance of which gives us a simple, unmixed sensation of color. These are pure red, which has no bluish or yellowish tint, pure green, yellow that is neither reddish nor greenish, and pure blue. These four primary colors form two pairs, red and green, yellow and blue. The two colors of each pair are called antagonistic colors, because they mutually exclude each other from exciting sensation in the retina. We can conceive of a blue which excites at the same time the sensation to some degree of green or of red, but not of a blue that produces the sensation of yellow.

Each color engenders the sensation of white in addition to that of its own hue in consequence of its varying brightness. This is because each color has a white "valence" in addition to its color valence, and both the brightness and the saturation of a color depend on the ratio between these two valences. The primary colors possess only one color valence along with the white valence, while the mixed colors have two. It is a physical necessity in the perception of colors that a physical accompanies the psychical act. Although we do not yet know the details of the metabolic changes in the retina, yet we can conceive with *Hering* those parts within the eye, the condition of which is intimately associated with the vision of colors, to be visual substance.

We may also suppose that each color corresponds to a quite fixed stimulation of a certain part of this substance, that color and stimulation are necessarily associated. As regards the nature of these stimulations of the visual substance I will quote a few words from *Hering*. "The nature of life lies physically in the metabolism of the living substance, by which, on the one hand, material is formed which is thrown off as something foreign, and on the other materials that are suited to it are taken up at the same time and formed into constituents of the living substance. The latter process has been called assimilation, an expansion of an old term used in the physiology of plants, and to this I have opposed the term dissimilation for the former process. While we can separate the conceptions of these two processes we cannot conceive of them as simply taking place side by side, and imagine the living substance to be a dormant mass which simply gives up waste material on the one side and takes up new material on the other. We have rather to think of assimilation and dissimilation as two intimately interwoven processes which constitute, in their peculiar way, the unknown metabolism of the living substance and take place at the same time in every minute portion of the latter, therefore this substance is nothing constant or quiet, but ever changing more or less in itself, ever active."





Rebman Company, New York.

All colors may be arranged according to tone in a socalled color circle. According to *Hering*, "If we choose a certain color in such a circle as the starting point, for example, a red like that with which a spectrum usually begins at its long waved end, we see the red constantly becoming more distinctly yellowish, while the redness of the color correspondingly decreases until we pass through orange and golden yellow to yellow which shows no trace of the red so plainly visible in orange. Other yellow colors follow this yellow in which green appears more and more (sulphur yellow, canary yellow). Farther on green becomes more and more distinct, as in sap green, while the yellowness becomes less marked and we finally come to a green that shows no trace of yellow. Following this green are other green colors in which blue may be perceived (water green), farther on the blue grows stronger, the green weaker (water blue), until we reach a blue that has no trace of green. To this blue succeed colors in which reddishness grows stronger and stronger, the blueness correspondingly decreasing (blue violet, red violet, purple red) until we come to a red from which the last trace of blue has disappeared. To this red succeed red tones with traces of yellow until we reach the red from which we started."

We may suppose, to use very popular language, that under the influence of light the visual substances are either assimilated or dissimilated. The white valences exert a dissimilative action upon the visual substance for the perception of black and white, while in the absence of light assimilation takes place and we have the sensation of black. If we take a red green, or a blue yellow, perceptive substance, pure red dissimilates the red green substance, while pure green assimilates it. If a pure red and a pure green are thrown on the same place in the retina it depends on the ratio between them whether assimilation or dissimilation prevails, and the resultant seasation is either red or green. If two antagonistic colors are used that are equal in their effect upon the visual substance their color valences are abrogated, the only effect that remains is that of the black and white perceptive substance, and we perceive only white of a certain degree of brightness. That the antagonistic, or complementary colors act in fact as opposing forces may be demonstrated with *Hering's* color top.

In order to understand color blindness we have to suppose, according to *Hering's* theory, that one or both colored visual substances are absent. If the substance perceptive of black and white is the only one present the color blindness is total. All colors then act only through their black and white valences and are therefore perceived only as white of varying degrees of brightness, or as gray.

In fact, the color of the entire world appears to the *totally color blind*, to be gray on gray. Such persons are easily recognized. They have a great deal of photophobia, their visual acuity is much impaired, and they usually have nystagmus. On the other hand their vision in the dark is decidedly better. The totally color blind reaches the optimum of adaptation in a few minutes. In order to obtain a conception of the vision of the totally color blind it is only necessary for one to picture to himself how he would see if he were to remain for a long time in the dark. Persons with total color blindness are seldom met with, and cases of yellow blue blindness are also very rare.

Red green blindness is of much greater practical importance. According to *Hering's* theory this is due to an absence of the red green visual substance. Two types of this anomaly are met with. In one the spectrum is shortened and the person has relative blue sightedness, in the other the spectrum is not shortened and the person has relative yellow sightedness. The presence of this anomaly is of great importance in many occupations. Persons so afflicted are excluded from the railroad service, as well as from various other callings. It is a condition that is widespread; it is met with in 4% of all men; women are less often affected. When we test the color sense we must be in a well lighted room, because the adaptation of the eye is of importance to the color vision. It is astonishing how well such a patient is able to name the different colors shown him. This is because he has been accustomed from childhood to notice very minute differences of brightness and saturation, and has learned to judge far better than we can what the color is from the difference in brightness. We can therefore consider no method of color testing as decisive in which the patient has to name the color. In *Adler's* test with colored pencils the patient is directed to write with each pencil the name of its color. The methods in which colored signals like those used on the railway are employed and the patient is told to name the signal are of just as little use, for most red green blind persons can distinguish red and green signal lights apart by their brightness. Yet an employee who could pass this test might occasionally mistake the signals and is therefore unfit for service. On the other hand it may happen that a man with normal color sense may name the colors wrongly, because he is not well acquainted with them. Hence it is wrong to ask a patient to name the various colors in a test for red green blindness. The only method that is unobjectionable is one in which the errors that the color blind commits in the confusion of colors can be perceived.

The so-called *Holmgren's* wools are used for this purpose. One of the variously colored skeins is taken as a test, and a person who is red green blind is apt to confound other colors with it. Red and green of the same brightness are distinguished by the red green blind by the fact that the former appears darker to him than the latter, but if we gradually diminish the brightness of the greens until the color appears to the red green blind to be of equal brightness with the unchanged red it will be difficult for him to tell the two colors apart. We therefore pick out a skein and ask the patient to select from the mass of skeins that are left all those that seem to him to be similar to it. If we pick out a bright green skein, in the color of which there is neither blue nor yellow, the patient may place beside it gray and red brown, or rose colored skeins. Or we may choose a purple that is neither bluish nor yellowish, and then those patients who belong to the first type will pick out the darker violet colors, while those who belong to the second type will take the brighter shades. Our attention is often attracted to the anomaly by the way in which a man with red green blindness behaves, by the slowness with which he sorts the colors.

We have other methods with which to confirm the diagnosis, one of which is the pseudoisochromatic test, in which we show the patient two objects that differ in color to the normal eye, but are confusing to those blind to certain colors. A good test is *Stilling's* tablet, which is composed of spots of various forms on a white background that are of the same tone, but differ in brightness and saturation. Between these spots lie spots of the confusion color which in combination with the former make figures and letters. The test shows whether the patient can read these characters or not.

The examination made in Germany is with *Nagel's* color tablet, which consists of a circle of little colored discs on a white background. On many of these tablets the circles are of only one color, but the discs vary in brightness and saturation. The color blind often thinks these circles are of different colors, because he takes the differences in saturation and brightness to be differences in color. On other tablets the circles are of various colors, and the color blind is unable to distinguish those that have only one color from those that have many, because the color tones chosen represent the confusion colors.

We can study the color blind more accurately by means of an apparatus like that devised by *Hering*, which is based on the fact that a perfectly valid color equation can be established for every person who is blind to two colors, which appear absolutely different to the normal eye.

203. Tonometry and Its Clinical Significance

In making purely scientific, experimental investigations we can introduce into the eye a canula connected with a manometer, in order to measure the intraocular tension, but this method is not available for clinical purposes on man, so we have to resort to an extraocular measurement of the tension.

In practice we rely mainly on digital palpation. We ask the patient to look down, place the tips of our two forefingers upon the upper lid as near as possible to the supraorbital margin, because the lid is thinnest at this place, and then make alternately with them a slight pressure on the sclera. In this way we are enabled to estimate the height of the intraocular tension from the amount of force we have to use in order to overcome the resistance of the capsule of the eyeball and to produce in it a distinctly perceptible impression. We designate the different degrees of tension determined in this way by T+1, T+2, T+3, T—1, T—2, T—3, as suggested by *Bowman.* But the results obtained in this way can represent no exact values, although the value of digital tonometry is not to be underestimated; it is clinically indispensable. Anyone who has a delicate hand and a well developed sense of touch can detect with certainty very slight differences of tension, but the sources of error in this method are very evident. In the first place we can obtain by it no values that can be expressed in figures, and secondly the unlike conditions of the eyelids, the variations in the intraorbital cushion of fat and the imperfection of the sense of touch as well as of the pressure of the finger tips, render difficult an exact determination of the tension. It is particularly hard to get in this way an idea of the fluctuations of tension that may take place. It is therefore easy to understand why efforts have been made for a long time to supplant digital tonometry by means of instruments. The best tonometer for clinical purposes of the large number that have been devised is that of *Schioetz.* I will read from the directions that accompany this instrument. "Before using this instrument the operator must see that the indicator is correct by placing the apparatus upon the convex model that is furnished with it, when the needle should point to 0[1], the first division of the scale. The measurement is commenced with the 5.5 gram weight. When the eye that is being examined has a normal intra-

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[[1] The zero mark is supposed to indicate the first line at the extreme left end of the scale. Its presence has proved rather confusing and apt to lead to an erroneous interpretation of the readings, so it has been omitted on many of the instruments.—*F.*]

ocular tension the needle points to between 3 and 6 mm. When the tension of the eye is increased to a considerable degree the needle may be directed to 0, or even a little to the left of the scale; the weight must then be changed until the needle shows a proper deflection. When the needle points to 0.5 or to 1 a heavier weight must be used. When the weights used are 7.5, 10, or 15, the deflection must be not greater than 4, or at the most 5; when the deflection is greater than this a lighter weight must be used. Deflections of from 2 to 4 mm give the most satisfactory results. The examination is made most conveniently when the patient is lying on a bench, or better, on a high operating table with his head bent rather backward, so that his eyes look directly upward.

"The eye or eyes are then to be rendered anæsthetic with a drop of a 2% solution of holocain. The burning that follows the instillation lasts a few seconds and as soon as this has passed away the eye is anæsthetic and ready for measurement. The patient is directed to look straight upward, the examiner carefully separates the lids with one hand without making any pressure upon the eyeball, and places the apparatus on the centre of the cornea with his other hand. The outer cylinder is then pressed down a little, so as to allow the instrument to stand alone and to press with its own weight simply upon the eye. The indication of the needle upon the scale is then read. The reading is done most easily by an assistant, but the examiner can do it himself if the scale is turned toward him. Three measurements should be made and an average made of the readings. The diagram indicates the number of millimetres of mercury that correspond to the position of the needle. The four curves correspond to the weights, 5.5, 7.5, 10, and 15."

The studies of *Schioetz* have informed us that the intraocular tension in the normal eye may vary between 16 and 27 mm of mercury, but the great clinical value of the apparatus is due to the fact that it enables us to form a judgment as to the actual state of affairs in pathological conditions, especially when the tension is increased. The use of this instrument should never be omitted in any case of increased tension, for it will certainly contribute to enlarge our knowledge concerning the nature and course of glaucoma.

The importance of an exact determination of the intraocular tension may be learned from the following case, in which the action of meiotics upon the pathologically elevated tension will be demonstrated. The patient is a young girl with chronic glaucoma. There is a deep glaucomatous excavation of each optic nerve. Iridectomy was performed some time ago on her left eye with the result that the tension was reduced almost to normal, but the field of vision contracted very rapidly after the operation to the point of fixation. The field of vision of the right eye has become greatly contracted in the meantime, so the question that we have to decide is whether an operation should be performed on this eye, or whether it is not advisable for us to confine ourselves for the present to the use of meiotics. To justify such a conservative course of treatment a careful clinical observation must be made to decide whether meiotics are able to reduce the pathological pressure to normal and to keep it at its proper level.

We begin the examination with the instillation of a drop of holocain in each eye, determine the tension before the instillation of eserin, and then ascertain how the tension is influenced by the meiotic. The result of this experiment is shown in the following curve.

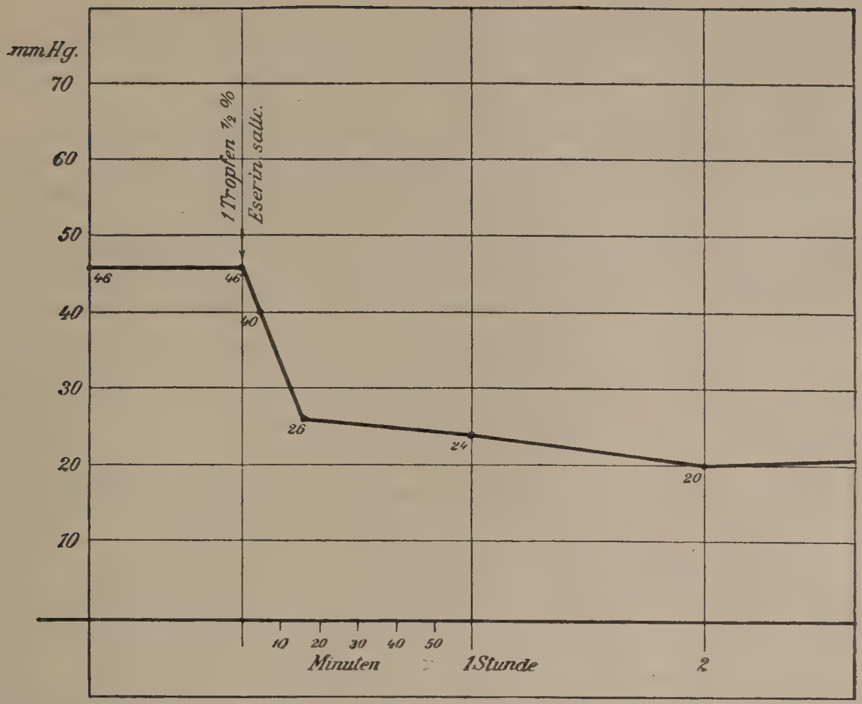

FIG. 186.—Influence Exerted by Eserin upon the Tension of a Glaucomatous Eye.

204. The Detection of Simulation

It sometimes happens that patients make incorrect statements in the functional tests of their eyes. When we have to deal with such patients we must first determine the exact condition objectively and then the lack of harmony between the result of the functional test and the objective condition may lead us to suspect simulation. We have first to decide whether the patient is trying to simulate a disease, or a functional disturbance of the eye, and then take measures accordingly.

The first group of malingerers who simulate diseases of the eye may be dealt with quite briefly. It often happens that a person who has been hurt rubs his injured eye with his finger, or with a cloth, before he comes to the clinic, in order to simulate a greater degree of irritation. Others produce a conjunctivitis artificially by placing foreign bodies, such as dust, ashes, snuff, gypsum, or meal, in the conjunctival sac. Even such strong irritants as soap suds and tobacco juice, and even such caustics as nitrate of silver, corrosive sublimate and sulphate of copper are used for this purpose. When we suspect a conjunctivitis to be artificial we examine the secretion in the usual manner. The greatest change in the conjunctiva ordinarily is to be found only on the lower lid, because this portion is the most accessible to the laity.

This traumatic irritation of the conjunctiva is caused to disappear most quickly when we apply a firm dressing under a bandage, a form of treatment exactly opposite to that which we ordinarily use. Self mutilations of the cornea and lens we do not meet with in this connection, but a favorite method of simulating disturbances of the pupil and accommodation is to instil atropin. In these cases also the isolation of the patient and the application of a firm dressing reveal the true state of the case very soon through the passing off of the mydriasis. A simulated error of refraction, particularly nearsightedness, is detected immediately by skiascopy, or by the examination of the upright image with the ophthalmoscope. School children are particularly apt to simulate myopia to make themselves interesting.

Diseases of the lids may be simulated in cases of accident. Blepharospasm especially is apt to be exaggerated, and a ptosis is occasionally simulated. Many patients complain that an eye that has been injured still lacrymates and is photophobic when it is perfectly pale. As we must in no case be unjust to the patient it is well to keep such an eye in a dark room for a long time and then bring it into the light. Eyes that are still sensitive after an injury are apt to redden and lacrymate under such circumstances, even though they are ordinarily pale.

Patients sometimes complain that after the injury of one eye the vision of the other is bad. This may be the case when the second eye was not used before the injury in consequence of some error of refraction, and is then forced to work, perhaps with a glass to which it is not accustomed. It is not difficult to bring out the actual facts in all of the examples that have been mentioned in which pathological conditions are exaggerated, or simulated.

The simulation and aggravation of functional disturbances is of more interest. The most important forms met with in practice are:

1. Simulation of blindness of both eyes.
2. Simulation of blindness or of amblyopia of one eye.
3. Simulation of amblyopia of both eyes.

I. Simulation of Blindness of Both Eyes

The reflex reaction of the pupil to light is first tested. A prompt reaction in both eyes cannot of itself prove the alleged blindness of both to be simulated, for the pupillary reaction may be preserved when the visual centres in the occipital lobes have been destroyed, but cases of this nature are rare and they are characterized by other symptoms. A prompt reaction of the pupils to light in a man who is otherwise well, but claims to be blind in both eyes must arouse the suspicion that he is simulating. On the other hand a bilateral absence of the reaction to light in no way indicates double amaurosis. I need only to refer to the reflex immobility of the pupil in the early stages of tabes. We simply take a patient who simulates blindness of both eyes into the clinic for some time and observe his entire behavior, for it is impossible for one who sees to counterfeit continuously the actions of a man who is truly blind. If we bandage his eyes the simulation will be disclosed very soon, because he will move about in a manner entirely different from that in which he moves when he is using his eyes, and if we tell him that the condition will improve under this treatment it is not apt to be long before improvement takes place.

II. Simulation of Blindness of One Eye

Blindness of one eye is simulated more often than total blindness. In such a case likewise we usually find the test of the reaction to light to be decisive. When an eye that is alleged to be blind shows a direct reaction of the pupil to light we know that it is sensitive to light, for those cases in which a direct reaction is present in spite of unilateral amaurosis are so extremely rare that they scarcely need to be taken into account.

If the pupillary reaction is good and suggests simulation we test the fixation of the eye that is said to be blind. For this purpose we may ask the patient, as suggested by *Schmidt-Rimpler*, to hold out his finger and to direct his eye at it. The blind man does this because he knows by feeling the position of his hand, especially if the examiner presses upon it a little with his finger, but the malingerer is very apt to look in some other direction, and this brings to light his insincerity.

We can also use the following test with a prism to determine absolutely whether the eye can fix. We cover the good eye and ask the patient to look straight ahead toward a candle with the one that is said to be blind; then we suddenly place a strong prism with its base up or down in front of the eye. The image of the object of fixation is thereby displaced toward the apex of the prism, and if the eye is a seeing one it immediately makes a corresponding reflex movement, which proves that it sees and fixes.

We can also demonstrate binocular fixation in such cases by the method suggested by *Alfred Graefe.* We let the patient look quietly at a light with both eyes and then place a prism with its base in or out in front of the one that is said to be blind. The double images caused in this way excite fusion at once and the eye immediately makes a corresponding movement of adjustment toward the apex of the prism. If the eye performs this movement and returns to its original position when the prism is taken away it both sees and fixes. This movement of adjustment will not take place if the eye is amblyopic. Another method of testing binocular vision is to bring an object close to the eyes in the middle line; if one eye is blind or amblyopic it soon deviates outward; then if we cover the fixing eye and the deviating eye makes a movement of adjustment we know that it can see.

It is a very good idea to place a prism with its base up or down before the eye that is said to be defective, when the case is suspected to be one of simulation of amaurosis or of a high degree of amblyopia of one eye, and have the man walk downstairs; if he is simulating he will have difficulty because of the double images he sees.

III. Simulation of Amblyopia of One or Both Eyes

A higher or lower degree of amblyopia is simulated more often than total blindness of one or both eyes. When the simulation is that of amblyopia of one eye we use tests in which we deceive the patient as to whether he is looking at the object with his good eye, or with the one that is alleged to be amblyopic. These tests can also be used in cases of simulated blindness of one eye. Under ordinary conditions we are unable to tell what we perceive with the right eye and what with the left. Our binocular estimate of depth depends on the blending of different sorts of visual impressions. This is shown most easily by looking at a reflecting, lustrous object; it is impossible to say for which eye a certain place looks bright or dark. There is, however, one precondition necessary to success in catching malingerers by this method: the eye that is said to be amblyopic must have equally as good vision as the other, for otherwise the malingerer notices the sharpness of the images that correspond to each eye. When the vision of one eye is actually not as good as that of the other the vision of the latter may be made worse so as to correspond with that of the former by the interposition of smoky gray glasses. The following tests may be made systematically one after the other.

First we take a visual impression of one and then of the other eye. *Wessely's* modification of *Herter's* test is to place two candles in the dark room, one of which is made to appear red and the other green by the interposition of glasses; then the light from the red and then the green candle is thrown by a plane mirror without regularity into the one eye and into the other, and the patient is directed to state which color he sees. Next we ascertain whether the eye that is said to be bad has light perception by the use of the double images produced by prisms, in the way suggested by *Alfred Graefe.* We hold a square prism of 12° with its apex down in front of the good eye in such a way that its base crosses the centre of the pupil horizontally and so causes monocular diplopia, the upper image of which corresponds to the true position of the light, while the lower is produced by the deviating action of the prism. The patient has to admit that he sees double with his good eye. Now we move the prism up so as to entirely cover the good eye and the patient continues to see double, the lower image produced by the prism remains in the same place, while the upper image, which previously was seen by both eyes is seen now by the second eye alone; hence if the patient continues to see double he must see with the eye that is said to be bad.

In order to determine accurately the degree of vision present in the eye in which amblyopia is simulated we occasionally render the test object invisible to one eye through the interposition of glasses. For this purpose we use tests in which the patient is deceived as to the size of the test object. For example we may use test charts on one of which all the lines of letters are one number smaller than on the other; or we may have him read from types which we remove farther and farther from him, continually substituting larger ones, and we can often prove in this way that he has been able to perceive much smaller characters than the smallest that he claimed to be able to see on the test chart at the same distance.

One test by means of which many malingerers are detected is to have the patient read the characters on the test chart at the distance of 5 metres; if he cannot read below a certain line he is caused to hold the test chart in front of his chest, is placed in front of a mirror at the distance of 2½ metres, and told to read the characters in the mirror at half the original distance. Of course the distance of the test is still 5 metres, in consequence of the reflection in the mirror, but many malingerers who do not know this read twice as many lines.

Another way in which we can frequently ascertain the degree of visual acuity of the eye which is alleged to be bad is to place a convex glass of 3 or 4 dioptres in front of the good eye, if it is known to be emmetropic, while the patient is looking at the ordinary test types, i.e., looking at a distance. This impairs the vision of that eye so that it can no longer perceive even the largest letters. If, in spite of this, the patient reads several rows he must be able to see them with the eye that is said to be bad. In testing near vision we place inversely a concave glass before the good eye that is so strong that it cannot be overcome by the accommodation; the reading must then be done with the eye that is said to be bad.

The interposition of cylindrical glasses, as suggested by *Jackson,* is still more successful. We place a plane glass before the eye that is said to be bad, and before the good one a combination composed of a concave and a convex cylinder of the same strength with their axes in the same direction, so that the effect is that of a plane glass. The patient finds that he can see very well at a distance with these glasses. Now we busy ourselves with testing the alleged bad eye with glasses and manage to rotate one of the two cylinders about 90° without attracting the attention of the patient; in this way the image will be so distorted that the good eye is excluded from the act of vision. If the patient continues to read he must do it with the eye that he claimed was bad.

The test object may also be made invisible to one eye by the use of colored glasses. Colored glasses allow only a certain part of the colors of the spectrum to pass through them and obliterate the rest, so *Haselberg* constructed test types printed in colors for this purpose. Each letter is so composed of black and red that the black part forms a complete letter and the red piece added changes the letter to another one, e.g., a P to a B or an R, so that the letters appear to be one or the other as they are seen through a green or a red glass. We place the red glass before the good eye, the green glass before the other, and then if the patient reads the complete letters we know that he is malingering and proceed at once to determine the visual acuity of the eye that is said to be bad.

The same principle may be carried out in another way. We may write the name of the patient wrongly with red crayon on white paper, place a red glass before his good eye, a green glass before his other and ask him if that is the right way to write his name. The red glass obliterates the writing with red crayon, so if the patient reads the name he is convicted of simulation.

It is seldom necessary to render the good eye incapable of doing near work by means of atropin, but when this has been done, if the patient is still able to read print at the proper distance he can do so only with the eye that is said to be bad.

The test recommended by *Javal* is simpler. If while the patient is reading a pencil is held between his eyes and the book, not too close to either, it will cover different portions of the print for the two eyes, but all the letters can be read because one eye sees those that the other does not, so if the malingerer reads the entire line he must do it with the aid of both eyes. The stereoscope may also be used to detect simulated blindness and amblyopia of one eye, because two different images are blended together by it. Instruments have also been devised which by a decussation of the visual lines render objects on the left side visible only to the right eye, and those on the right only to the left. The malingerer thinks he sees objects on the right side with his right eye and those on the left with the left, so he suppresses the image seen with his good eye and betrays himself by doing so. Such a crossing of the visual lines is obtained by *Graham's* tubes, modified by *Koster*. *Worth's* amblyoscope may also be used for this purpose.

IV. Simulation of Amblyopia of Both Eyes

The most difficult cases of simulation to detect are those in which there is reason to suppose that the vision of both eyes is impaired, but that the visual disturbance is exaggerated. The objective condition is primarily decisive, so we choose from the tests that have been described the ones that seem to be the most suitable for the case.

V. Simulation of Disturbances in the Field of Vision

Malingerers are very apt to simulate defects in the field of vision as well as impairments of the vision itself. If we test such a person with the perimeter we usually obtain a very small visual field. The imposition can be detected very readily by testing the field at various distances, as recommended by *Schmidt-Rimpler.* We place the patient beside *Hirschberg's* table and ascertain the limits of the field of vision at different numbers of metres and then the extent of the field of vision must be proportional to the distance in each case. But the malingerer is very apt to state that the field remains of the same size, or that it grows smaller as the distance increases. Most cases of simulation may be unmasked by this test. It fails only in the concentric contractions of the field in hysteria and traumatic neurosis, because in such cases the tubular form of the visual field is maintained even in this campimetric measurement. Traumatic hysteria and neurosis play an important part in accident cases, so that frequently we are obliged to recommend an examination by a neurologist.

APPENDIX

Asthenopia, or Eye Strain

By Matthias Lanckton Foster, M.D.

The great majority of patients, in this country at least, who seek the ophthalmologist complain of one or more of the symptoms that are loosely grouped together under either the term asthenopia or, one that is perhaps more accurately descriptive, eye strain. These symptoms are headache, a strained feeling in and above the eyes, which sometimes augments to severe pain, fatigue, burning and smarting of the eyes, blurring of the vision, blepharospasm, lacrymation, photophobia, nausea, vomiting, vertigo, less often insomnia, mental depression, nervous prostration, and, according to some writers, such reflex disturbances of the nervous system as chorea and epilepsy. They can hardly be said to form a symptom complex, because few if any patients present all or even a majority of them, and the presence of several is not necessary to the diagnosis. It often happens that persistent headache is the only one complained of, and that the patient has been referred to the ophthalmologist by a physician who has exhausted all the means at his disposal in a vain effort to locate its cause. Other patients present two or more symptoms combined in an endless variety of ways. The enthusiasm with which we have pursued the study of this subject has been a matter of ridicule in other countries, and it is very likely that we have made errors; but, if it is true that the commonest affections should receive the greatest amount of attention, we can console ourselves with the reflection that in this respect at least we have done right; that we have demonstrated and tried to overcome the difficulties which beset the path of anyone who deals with eye strain, and that the sufferings of many human beings have been mitigated or permanently relieved as the result of our efforts. Many questions are still unsolved and form subjects of lively debate. Advance in medicine has always been made through the efforts of such enthusiasts as those who maintain one side or the other in this wordy warfare; it is due to the study of such as they that we know far better how to relieve asthenopic symptoms than we did a generation ago, and it is to them we look for further progress in the future. Into these questions I shall not enter.

The first essential to success in handling the ever varying problems that confront us is accurate observation of the mechanical conditions with which we have to deal; the second is the careful elimination of such mechanical faults as are physiologically compensated for and may therefore be termed in a sense physiological.

Three forms of asthenopia are ordinarily recognized: accommodative, due to errors of refraction and to strain of the ciliary muscle; muscular, due to an abnormal strain of the extrinsic muscles of the eye; nervous, due to a fault in the nervous system. To these I am inclined to add a fourth: reflex, due to a morbid condition in the organism outside of the eye and nervous system.

Asthenopia Due to Errors of Refraction

One of the most difficult things to understand in the study of the effect of errors of refraction upon the human organism is the widely varying results produced by the same error upon different individuals. Most eyes are very tolerant; most people have demonstrable errors of refraction of which they are unconscious; many of those who become conscious of such errors remain comfortable when the spectacle frames are bent, when a spherical glass is tilted so as to produce an astigmatic effect, or when the axis of a cylindrical lens is altered several degrees, and patients with such tolerant eyes obtain satisfaction from anything like a fair approximation toward their true correction. This fact has had great weight in perpetuating the idea that accuracy in the fitting of glasses is not of such great importance after all. But many eyes, few in comparison to the great total yet many in number, are intolerant to even a very slight error of refraction, or to a little variation of the correcting lens from its proper position. Patients with such eyes are apt to complain of headache not associated with any symptoms which can be termed distinctly ocular, can be persuaded with difficulty to have their eyes examined at all, and yet are often completely relieved by a very weak glass, especially if the error is astigmatic. Such a case was that of an intelligent, successful business man who came to me with the positive statement that he had never had any trouble with his eyes and knew there was nothing the matter with them, but had come to have them examined purely to satisfy his physician. For several years his headache had been so bad in the morning that he could not dress without lying down two or three times to rest his head. After breakfast it became rather better, so that he could attend to his business all day, although it was always present. In the evening he would retire to a dark, quiet corner and sit there till bedtime. I prescribed +.25 cyl. axis 90° for each eye. Two days later his wife and family called to thank me for the first evening's social pleasure with the husband and father they had enjoyed for two years. A month later he told me he had not had a headache since he began to wear the glasses. From the reports of his physician I have reason to believe that the relief was permanent. The only symptoms presented by this patient were headache, seemingly not connected with the eyes, and perhaps a slight degree of photophobia, evidenced by his desire to sit in a dark place during the evening. Other patients have symptoms that appertain more distinctly to the eyes. The only way in which such minute errors of refraction can be excluded as the cause of the asthenopia in any particular case is by having the patient wear the correction for a while; if it is not the cause the glasses will be abandoned quickly because of the incidental annoyances.

Accuracy in the determination of the error of refraction can be attained in many cases either with or without a cycloplegic, in some it cannot be secured without, and in a comparatively few the results obtained under a cycloplegic are misleading, but in every case painstaking effort on the part of the physician is essential. The glasses must be worn while the eye is in its normal state of physiological activity and no way has yet been discovered by means of which to measure that activity in an objective manner in the individual eye, so that it may be fitted scientifically without asking the patient any questions. Until such a way has been found the subjective test must remain the court of last resort, for by it alone can we determine how far and in what way to modify the glasses indicated by the objective examination.

The first examination should always be made without a cycloplegic. We test the vision of each eye separately, and thereby gain much information, as has been shown in the text. We learn from the ophthalmometer the corneal astigmatism, though not the total, and the probable axis of the correcting cylindrical glass, and a glance into the eye with the ophthalmoscope gives us a fair idea of the nature of the refractive error. Now we know, almost with certainty, whether the eye is hypermetropic or myopic, whether much astigmatism is present and, if it is, about its degree and axis. Little if anything more can be learned from skiascopy without a cycloplegic, for at best it gives us only an approximate idea of the refractive condition, because of the activity of the ciliary muscle, and at worst it is confusing. The information we have thus obtained reduces the choice of glasses to a very few combinations. If, for example, the vision of the eye is 6/7.5 the corneal astigmatism .50 D with the rule, and the ophthalmoscope reveals about 2 D of hypermetropia, we may rest assured that we have the right glass if the vision is brought up to 6/6 or better by a +1 D or a +1.50 D spherical with or without the addition of a very weak cylindrical, and the patient is made comfortable by that correction. But if perfect vision can be obtained only with the aid of a concave glass, i.e., if the subjective test does not agree with the objective findings, we have positive evidence that too strong an innervation is sent to the ciliary muscle and that the use of a cycloplegic is necessary to do away with its abnormal contraction and to allow us to measure the refraction of the eye with the accommodation eliminated. When dealing with a child I usually prescribe a cycloplegic immediately after the preliminary examination, because the objective test is almost certain to be vitiated by the energetic contraction of the ciliary muscle and the subjective is apt to be unsatisfactory.

We must remember in the subjective test not only to choose the strongest + glass in hypermetropia and the weakest — in myopia with which the best vision can be obtained, but also to ascertain whether any further improvement can be obtained by the addition of a very weak cylindrical lens at any axis. This is not being finical, it is simply being accurate in observation. After each eye has been examined the glasses are adjusted so that each eye will see through the centre of the correcting lens and both are tested together. The patient is directed to use his eyes and to state whether the glasses make them and his head feel more comfortable or not. The comfort may sometimes be made perfect by a slight change in the strength of the spherical, or in the axis of the cylindrical glasses. In hypermetropia the strength of the spherical occasionally needs to be increased a little, but it happens in rare cases that the full correction of the manifest hypermetropia cannot be worn with comfort. Thus, a woman who obtained the best vision of each eye, 20/15, with a +1 D glass was unable to wear anything stronger than +.50 D with comfort. I could not discover the reason, though several examinations were made in the course of two or three months.

When the glasses selected in this way do not correspond with the objective findings, or when they are not comfortable, a cycloplegic should be prescribed, in the first case because it is evident that the measurement of the refractive error has not been accurate, in the second because either the measurement is incorrect or the asthenopia depends on some other cause, and the chance of a mistake must be guarded against. Many good refractionists believe that the use of a drug to paralyze the accommodation is essential in all cases if we would measure the refraction accurately. They would be right if no source of error were introduced by cycloplegia, which is physiologically and functionally an abnormal condition of the eye, and if the asthenopic symptoms could not be relieved otherwise, but a glass can hardly be pronounced incorrect that corresponds to the objective condition of the refraction determined in the manner described and gives relief to the subjective symptoms. I have repeatedly used cyclopegics for a further test of such patients and found them of no value whatever, while I have found them of great value when the subjective did not agree with the objective test, or when the glasses did not give relief. A point which seems to be of importance in this connection is that the astigmatism is not always the same, either in degree or axis, when the eye is under the influence of a cycloplegic as it is when the organ is physiologically active, in the condition in which it is to be used, and it is the astigmatism in the latter condition that needs particularly to be rectified.

A point worth noting is that if the symptoms disappear when the ciliary muscles are paralyzed, the probability is increased that the asthenopia is due to an error of refraction.

It is when the eye is under the influence of a cycloplegic, not simply a mydriatic, that skiascopy performs its perfect work. Then the refraction can be measured with absolute accuracy by means of the skiascope down to .12 D of hypermetropia, myopia, and astigmatism, while the axis of the correcting cylindrical lens can be determined positively. In this country we use the plane mirror in preference to the concave, so the movements of the shadow to which we are accustomed are in exactly opposite directions to those given in the text. The movement is with the light on the face in hypermetropia, emmetropia, and myopia, with the point of reversal behind the observer; in the opposite direction in myopia, with the point of reversal in front of him. This is because the rays of light are reflected from the plane mirror as though they came from a point as far back of the mirror as the source of light is in front of it. When the light coming from the pupil seems to move in the same direction as the movement of the mirror the observer is between the eye and the point of reversal, and a plus lens is to be found which will stop all apparent movement when it is placed in front of the eye. The rays of light which emerge from an eye that is emmetropic under this condition are parallel and will be made to converge to a point 1 metre distant by the interposition of a +1 D glass, so if the observer keeps his eye 1 metre from that of the patient he needs only to subtract +1 D from the number of the glass needed to suppress all apparent movement of the light in the pupil to learn the true refraction. This is exemplified in the text. To measure the astigmatism we ordinarily use spherical glasses, determine the refraction in one meridian and then in the opposite. The presence of astigmatism in the eye may be recognized from the presence of a bright band across the pupil, which may be seen at once if the astigmatic error is greater than any other, but is not apt to be noticed otherwise until after a spherical glass of sufficient strength to render the astigmatic error the greater has been placed before the eye. The axis of the astigmatism is indicated by this band and may be determined accurately by means of the axonometer, devised by *Thorington,* which consists of a black metal disc as large as an ordinary trial lens, with an aperture 12 mm in diameter in its centre and two heavy white lines, one on each side, that pass from the circumference across to the opening in the centre so as to bisect the disc. The axonometer is placed in the trial frame so that the cornea occupies the central opening and is turned slowly during the examination until the band of light joins the two ends of the two white lines so as to form one straight line. The arrow head at the end of the white line points to a degree mark on the trial frame which gives the axis of the correcting glass.

Regular astigmatism of all varieties may be measured readily and accurately in this way. Irregular astigmatism, whatever its cause, presents many difficulties; the only practical way to deal with it is to apply the general principles of skiascopy as well as we can to each individual case. A curious phenomenon sometimes seen is the scissors movement of two bands of light which approach and separate as the mirror is moved back and forth in a certain meridian. It indicates a form of irregular astigmatism in which the refraction in a certain meridian differs in one portion of the pupil from what it is in another, and is caused by an obliquity of one or more of the dioptric surfaces, usually of the crystalline lens. It may be obtained from most eyes by casting the light with the skiascope so that it will enter the pupil at a considerable angle to the optic axis, and from the artificial eye of the manikin by placing the lens that represents the dioptric surfaces so that the light will pass through it obliquely.

A phenomenon that is apt to be confusing to the student is the spherical aberration induced by rays of light which pass through the peripheral portions of the cornea and lens. This may be either positive or negative. In positive aberration the peripheral refraction is stronger than the central, i.e., the rays of light that enter the pupil through the periphery are brought to a focus sooner than those that enter through the centre; in negative aberration the central refraction is the stronger. When we measure the refraction of the eye the play of light and shade in the peripheral portion of the dilated pupil has to be disregarded, for these peripheral rays of light are intercepted by the iris under normal conditions, and we have to concentrate our attention on what takes place in the centre, because that portion alone is to be taken into account which comprises the visual zone when the pupil is of normal size. Although the expert can disregard these aberrant rays to a very great degree they form a possible source of error which needs to be guarded against by a postcycloplegic examination. A high degree of negative aberration suggests the presence of conical cornea.

After the refraction under a cycloplegic has been measured with the skiascope the findings should be compared with the results of the first examination and the variations noted. If the only difference is a slight increase of the hypermetropia, no more than would normally be rendered latent by the natural tonic contraction of the ciliary muscle, taking into account the age and general physical condition of the patient, and if the subjective symptoms were not relieved by the glasses found on the first examination, it is almost certain that the asthenopia does not depend on the error of refraction, but is due to some other cause. If other differences are present, such as a large increase of the hypermetropia, or a variation in the astigmatism, a final examination should be made some days later, after all traces of the cycloplegic have vanished from the eyes. The skiascopic findings may be proven correct of the cycloplegic eye, but it does not necessarily follow that they are correct of the eye in its physiologically normal condition, that in which it is to be used. For example, the astigmatism may be quite different after the mydriasis has passed away, possibly because of the influence exerted by the aberrant rays when the pupil is dilated, possibly because of other reasons. This is not the case, as a rule, but the possibility must be borne in mind. The skiascopic findings are usually of essential value in the final choice of glasses, but, like the other objective findings, they do not furnish all the needed information. We have still to learn in how far and in what way the refractive condition we have demonstrated objectively is modified by the physiological activity of the ciliary muscle and the exclusion of the peripheral rays of light by the normally contracted pupil.

To do this we place before the eyes the glasses suggested as probably correct by the skiascopic examination and change them in the interest of vision and comfort until the most satisfactory combination has been obtained. It is well to try each eye separately and then both together, though it is rarely that any change will be called for in the latter test. Sometimes the axes of the cylindrical glasses need to be altered a little, and then it may be well to examine for cyclophoria, in order that if it is present we may adjust the cylindrical glasses to meet its indications as well as to correct the astigmatism.

The principal source of the perplexities that arise during an examination of the eyes for glasses is the behavior of the ciliary muscle, which may render latent a larger portion of the total hypermetropia than corresponds to its normal tonicity, even to such an extent as to simulate myopia. The muscle usually relaxes sufficiently during an examination of the upright image with the ophthalmoscope to enable us to recognize with what we have to deal, but in rare cases we meet with a true spasm of the accommodation in which no such relaxation takes place. Such a spasm is usually obstinate and rarely yields to anything weaker than atropin. Even this drug seems inefficient at times. I have seen an apparent myopia persist in the eyes of a young man in spite of the constant use of atropin from June until September, and then pass off, leaving emmetropia. In this case I could detect no imbalance of the muscles, and was led to persist in the use of atropin because, although he seemed to be nearsighted when indoors, he could see a golf ball in its flight. In another case in which myopia persisted in spite of atropin the young man had about 3/4° of hyperphoria and the myopia disappeared after I had performed a partial tenotomy on his superior rectus in the hope of stopping his headaches. Two weeks later the muscular imbalance was the same as before operation, but was no longer productive of symptoms. The causes that produce these rare cases of true spasm of the accommodation are not yet known; the evidence is too insufficient and faulty for us to ascribe them offhand to mechanical faults in the extrinsic muscles or elsewhere. Other surprises await us. One thing is certain. Difficult problems are apt to arise which cannot be solved quickly and easily as long as we bear in mind the fact that the glasses are to be worn in front of eyes that are in the active condition in which Nature intended them to be used, and that they are also to repress abnormal activities in those organs, although glasses can be fitted in the majority of cases with little trouble. Each case must be studied individually, and then we find that in some the asthenopia is not due wholly, if indeed at all, to the error of refraction.

Asthenopia Due to Imbalance of the Extrinsic Muscles of the Eye

In a fairly large minority of the cases the symptoms depend on other causes, foremost among which is imbalance of the extrinsic muscles of the eye. One point needs to be made clear, that the presence of muscular imbalance is not per se any reason for intervention. Its presence can be demonstrated in very many persons who have no symptoms of asthenopia, and *also in many who have asthenopia from other causes.* In such cases the imbalance must be compensated for physiologically, or, as Professor *Roemer* terms it, must be physiological, or else it is itself a symptom of some other abnormal condition, when attention should be paid to the underlying cause.

When we have a patient who complains of persistent headache, or of any other of the symptoms enumerated as asthenopic, in whom we can find no error of refraction, or in whom the symptoms have not been relieved by correct glasses accurately adjusted and properly worn, an investigation of the muscular condition of his eyes is imperatively demanded. Time can be saved by making such an examination as a routine procedure in all cases, for occasionally the probability that the symptoms are caused by faulty muscular action is rendered evident at once, and sometimes the behavior of the muscles directs attention to the general condition and leads to an early recognition of a morbid condition elsewhere. It must be remembered that in very many cases we are dealing with tendencies to incoordination, with ocular symptoms of unbalanced nerve action, rather than with demonstrable lesions of the muscular or nervous system, and therefore that every condition of the organism which may disturb the nervous equilibrium, whether in the eye or not, is to be investigated and its bearing upon the individual case studied. An imbalance of the muscles frequently passes away, or is physiologically compensated for, when the glasses that correct an error of refraction are constantly worn, and it is well known that both mental and physical factors affect the nervous equilibrium and thereby the balance of the muscles; in such cases the heterophorias are symptomatic. But after all these cases have been excluded there remains a goodly number in which the imbalance is not symptomatic and not compensated for, but is a primary condition productive of trouble that needs correction.

The tests for imbalance of the ocular muscles may be grouped in three classes: 1, Those in which one image is displaced; 2, those in which one image is distorted; 3, those in which the image is neither displaced nor distorted. The prism test, first suggested by *v. Graefe*, is an example of the first class. This test can be made roughly and clumsily by holding the prisms in the fingers, but much more accurately, conveniently and quickly by the aid of the phorometer, which was devised by *Stevens*. This instrument consists of a base and upright rod to which is attached at right angles an arm furnished with a screw and spirit level by means of which it can be kept perfectly horizontal. Upon this arm is placed a frame containing a pair of 5° prisms which can be rotated together by means of a lever. The instrument is placed before the patient so that he looks with each eye through one of the prisms at a small light 6 metres distant. If we place one prism with its base up and the other with its base down the patient will see two images of the light, one vertically above the other, if he has binocular single vision and no tendency of the eyes to swing in or out; if the upper image is to the right or left of the lower a condition of exophoria or esophoria is present, and the deviation can be measured by the strength of the prism needed to bring it directly over the other. The strength of the prism needed to do this is found by rotating the prisms one way or the other until the images are in a vertical line, when the degree of exophoria or esophoria can be read from the attached scale. The amount of right or left hyperphoria is learned in the same way when the prisms are placed with their bases in.

The *Maddox* rod test is an example of the second class, the cover test of the third; both of these have already been described. But the cover test has an advantage that has not been mentioned—it is of subjective as well as objective value. If the patient has orthophoria he will see no movement of the object of fixation when the screen is moved because both of his visual axes remain directed at the same point when one eye is covered, but if he has heterophoria the object will seem to move, because the patient's eye has moved to regain the position of fixation. After a few trials an intelligent patient can describe accurately the direction and extent of this apparent movement. If it seems to be in the same horizontal direction as that of the screen he has exophoria, if in the opposite direction he has esophoria; if it seems to move downward when the right eye is uncovered, right hyperphoria is present; if it seems to move upward, the case is one of left hyperphoria. This apparent movement may be controlled by prisms and be measured exactly by them, as the degree of prism that stops the apparent movement measures the heterophoria. Some writers call this the parallax test to distinguish it as subjective from the objective observation of the eyes themselves, to which they refer as the cover test, but this distinction is hardly necessary, as we should note both the subjective and objective findings in such a test as this. The third class of tests seems to me the most trustworthy, but the others have strong advocates. All three can be used to advantage in many cases and the findings may be checked off against one another. The continual use of the prism test at least is able to train patients to reveal errors of muscle balance that do not exist. Some years ago I had a patient who had been examined with the prism test a great many times by a large number of physicians and could develop at will 15° of exophoria, or 15° of esophoria. The cover test revealed no movement of the eyes and no subjective movement of the object, so reliance on the prism test would have been misleading. This was an extreme case of the results that may be reached by too much training with prisms, but less marked ones are not so very uncommon and, unless they can be checked off in some way, the physician may be misled.

I think the duction test to be a very important one. By it we determine the ability of the muscles to overcome prisms with their bases out, in, up, and down, in other words, to adduct, abduct and sursumduct the eyes. The patient fixes his eyes on a small light 6 metres away; a square prism, with one edge resting on the arm of a phorometer, is held with its base out in front of one of his eyes, and as soon as the double images thus formed have fused it is replaced by a stronger one. This is continued until one is reached which the patient is unable to overcome so as to fuse the double images. The strongest prism overcome is the measure of the adduction of his eyes. The abduction is learned in the same way when the prisms are held with their bases in, the sursumduction when they are held with their bases up and down. Some writers call sursumduction with the base of the prism down supraduction, with it up infraduction.

Various instruments have been devised to do away with the inconveniences of this method of testing, among which may be mentioned *Risley's* rotary prism, and *Jackson's* rotary prism, but these seem to me to cause a certain amount of chromatic aberration and prismatic astigmatism which are not desirable.

The results of these duction tests are often of signal service. For example: if the phorometer shows a high degree of heterophoria the duction test should reveal a corresponding variation from the normal; if it does not, the heterophoria may be purely functional. Another point is that if the duction power is weak in all directions we are led to suspect a general nervous or muscular weakness due to some cause outside of the eye.

It is well to supplement the information thus obtained by a study of the field of fixation by means of the tropometer, another instrument devised by *Stevens.* It consists essentially of a telescope in which an aerial image of the cornea is formed near the eye piece, where a graduated scale permits every movement of rotation in any direction to be accurately measured. The patient places his head in the head rest so that the glabella and the commissure of the upper lip are in a vertical line, while his eye is fixed on the centre of the object glass, and grasps a wooden mouth piece between his teeth to keep his head immovable. He then moves his eye to the fullest possible extent in the four principal meridians, while the examiner notes the effect registered on the scale by the movement of the cornea. The approximate rotations thus obtained are given as: upward 33°, downward 50°, inward 48° to 53°, outward 48° to 53°. The rotations in all other directions are accomplished by the combined action of the muscles that make these four and can be neglected, as a rule. The line which connects these points when they have been found maps out the monocular field of fixation of the individual. This field varies in different persons according to the powers of their muscles and also according to their attention and effort, so moderate variations from the figures given, especially when they are proportionate, do not necessarily indicate any abnormality, but any marked variation, particularly if in one direction alone, should be carefully noted.

The perimeter may be substituted for the tropometer when the head of the patient is secured in position, as by a mouth piece, and a small electric light is moved along the arc as far as it can be fixed by the eye of the patient. If the examiner moves his head along the arc with the light he can watch the reflection of the latter from the centre of the cornea and notice its displacement at the instant the eye can no longer fix the object, and so the subjective and objective portions of the test can serve as checks upon each other.

The rotations of the eye determined in this manner are voluntary, those obtained otherwise are involuntary, and the results obtained in both ways should be studied together if we are to get an accurate knowledge of the behavior of the muscles.

A final test is for cyclophoria, which has been defined by *Savage* as the tendency of the vertical axes of the eyes to lose their parallelism with the median plane of the head. *Savage* says: "In the interest of binocular single vision this parallelism must be maintained by the oblique muscles, except in cases of oblique astigmatism. For this purpose there are four conjugate innervation brain centres: 1, The sixth conjugate centre sends an impulse to the two superior obliques to prevent divergence of the vertical axes when the point of view is in the extended median plane of the head, but below the extended horizontal plane; 2, the seventh conjugate centre sends an impulse to the two inferior obliques to prevent convergence of the vertical axes when there is to be cardinal fixation above the horizontal plane; 3, the eighth conjugate centre sends an impulse to the superior oblique of the right eye and the inferior oblique of the left to prevent torsion when the point of fixation is obliquely up and to the right, or down and to the left; 4, the ninth conjugate centre sends an impulse to the superior oblique of the left eye and the inferior oblique of the right to prevent torsion when the point of fixation is up and to the left, or down and to the right. Under the influence of one or another of these conjugate centres parallelism of the vertical axes of the eyes with the median plane of the head is maintained, regardless of the direction of the point of fixation. In a normal condition of all the extrinsic ocular muscles the obliques accomplish this purpose with ease. When conditions make it difficult for the obliques to maintain this parallelism, except when under an excessive nervous tension, there is cyclophoria. Plus cyclophoria means that the vertical axes of the eyes have a tendency from the median plane of the head; minus cyclophoria is a tendency of these axes toward the median plane."

This condition was first described in 1891 under the term "insufficiency of the obliques," and the name cyclophoria was suggested later as one that would conform to the nomenclature introduced by *Stevens*. Other names that have been used are plus torsion and minus torsion (*Maddox*), plus and minus declination (*Stevens*). The cause of a cyclophoria may be situated in the innervation centres, or in the muscles. Its presence can be detected, but it cannot be measured by means of a rotary prism if we use a straight line as a test object; the two lines seen will converge toward one end or the other if cyclophoria is present. It may be detected and measured by such an instrument as the clinoscope, also devised by *Stevens*, which consists of two hollow tubes, each of which has at one end a minute pinhole opening through which the eye can look, and at the other end a translucent disc on which is drawn a line, in the case of one tube from the centre straight up and in that of the other tube straight down. These tubes are so adjusted on a standard that they can be placed and maintained in the same horizontal plane which is indicated by a spirit level, but from end to end they can be directed horizontally or up and down. They can be made to converge or diverge to meet certain contingencies. The tubes rotate on their long axes and a pointer attached to each tube indicates on a scale the extent to which it is rotated. The small sight openings are so adjustable that the distance between them may be suited to the pupillary distance of different persons. For the accommodation of those who, on account of any high degree of refractive error, cannot see at the distance of the test objects from the eyes, there are clips in which refracting glasses may be placed. The tubes are adjusted so that the apertures in the eyepieces are placed exactly before the two eyes of the patient, that he may see the two lines simultaneously with each. He sees them as one straight line if he has no cyclophoria. If the line appears to be bent the tubes may be revolved about their long axes until it seems to be straight, and then the degree of plus or minus cyclophoria can be read from the scale.

When a condition of heterophoria has not only been found but has been established as the probable cause of the asthenopia we have to consider the form of treatment which is appropriate to the individual case. When the imbalance seems to be due to weakness of one set of muscles, such as the interni or externi, excellent results can frequently be obtained by gymnastic exercises of the weak muscles by means of prisms. Some physicians give nerve sedatives at the same time to diminish the abnormal contraction of the opponents of these muscles during such exercises, aud I think I gain some help from a very weak faradic current, one electrode placed on the conjunctiva over the insertion of the muscle to be treated, the other held in the hand of the patient, but the benefit obtained by this use of electricity has been doubted by many ophthalmologists. Such treatment as this, devoted to the strengthening of a weak muscle, is frequently advisable even when the heterophoria is not primary, but symptomatic of some trouble outside of the eye; it is sometimes of good suggestive value, but in such a case it should never be allowed to eclipse the need of searching out and if possible doing away with the underlying cause of the symptom.

When no such weakness of one set of muscles can be found, or when the attempt to strengthen them has failed, prisms may be prescribed for constant wear with their bases toward the muscles whose actions are to be favored. Such prisms allow the eyes to assume a position which is one of rest for them, and they relieve the symptoms in many cases. As a rule, it is not wise to prescribe a prism which will correct the entire heterophoria, it is better to give one that will allow the eye to assume a position in which the symptoms indicative of strain are relieved. It is not right to say that the strain must persist as long as the balance of the muscles is not mathematically perfect. We know that physiological compensation exists in many men who have muscular imbalance because the condition is not productive of symptoms, so it is not unscientific to suppose that in other cases a portion of the imbalance is compensated for, although we cannot determine instantly to what extent this is true in any particular case. If the correction of any portion of the demonstrable hyperphoria, exophoria, or esophoria, gives comfort and relieves the symptoms it has induced, this is sufficient. It is best to begin with an undercorrection and to feel our way up until comfort is secured, or the entire imbalance has been corrected. We can sometimes secure the desired prismatic effect by decentering the glasses used to correct the refractive error. As many prism degrees of effect can be obtained by decentering a spherical lens 10 mm as there are dioptres in the strength of the lens.

Cyclophoria is to be treated by a suitable adjustment of cylindrical glasses, by exercise of the obliques by means of such an instrument as the clinoscope, or by rotating cylindrical glasses in front of the eye. The advisability of operative intervention is, to say the least, questionable. It has been suggested that very delicate operations may be performed on the straight muscles to correct obstinate cases of this condition, but the suggestion has not yet met with general acceptance. One thing, however, seems to be certain: no operation should ever be performed on the obliques.

When all other measures have failed to give relief in heterophoria operative intervention is to be considered. The surgeon has to choose between a tenotomy of the overactive muscle and an advancement of its antagonist; this choice must be made in accordance with the demands of the individual case. The aim should be to obtain an undercorrection, but one that will relieve the patient. If no relief is obtained by a considerable decrease of the muscular deviation it seems illogical to persist in trying to get an exact balance by means of operations repeated many times, which sometimes deprive the eyes of the power of binocular fixation. It is better that the question should be raised again whether the asthenopia is not due to some other cause.

Asthenopia of Nervous Origin

The intimate relations that exist between the eye and all other parts of the body cannot be emphasized too strongly. The connection between the eye and a viscus situated in the abdomen through the nervous, circulatory, and lymphatic systems is just as close as that between any other two separate organs; the eye reacts to troubles situated in distant parts, and other organs react to troubles in the eye. We may have nausea and vomiting caused by ocular derangement, and we may have the symptoms of asthenopia produced by an indiscretion in eating. It is difficult to draw a hard and fast line between an asthenopia that may be called nervous and one which may be termed reflex, for both are symptomatic of trouble in some portion of the organism outside of the eye which is productive of nervous irritability. We may say that the asthenopia due to menstrual disorders is nervous, or we may call it reflex, and confine the term nervous to those forms which are caused by trouble in the nervous system alone. I prefer the latter. A border line condition is the asthenopia caused by anæmia, which is often symptomatic of morbid organic conditions, but when the irritation of the nervous system is due primarily to the impoverished condition of the blood the asthenopia may properly be called nervous. We meet with this condition frequently in convalescents who are much debilitated.

Persons suffering from over work, over worry, and lack of rest, often complain of asthenopia. When an energetic hard worker says that he has to drive himself to work which he previously enjoyed, that he dreads it and has to continually spur himself on to perform his daily duties, and when such a man has asthenopic symptoms that cannot be accounted for by his refractive condition, he needs a rest, for he is threatened with a nervous break down and the asthenopia is only one of the symptoms of the general nervous irritation. In such a case the symptoms are of fairly recent date, have been steadily progressive, and can, with a little trouble, be traced to their origin. When one is very tired a slight incoordination may quite well appear in the ocular muscles, so the test for muscular imbalance is apt to be unreliable and misleading.

Nervous asthenopia may be a symptom of a grave nervous disease. *Hansell* and *Reber* report a case in which asthenopia with hyperphoria seems to have been the first symptom of tabes, with the reflex immobility of the pupil the second. The hyperphoria passed away of itself within a year. It occurs in neurasthenics, when, for want of something better, we may ascribe it to a lack of nerve tone. It is also met with in hysteria. Sometimes the symptoms have been supposed to be due to a condition of anæsthesia or hyperæsthesia of the retina.

Occasionally asthenopia can be traced directly to smoking tobacco. I know a gentleman who cannot smoke a single cigar without suffering the next day from red, burning eyes. Another, whose smarting eyes I had tried in vain for some time to relieve, smoked five or six cigarettes a day. At the end of a week of abstinence his eyes were well; after smoking as before for a week they were as bad as ever; abstinence again for a week resulted in a perfect cure. The explanation is probably the toxic irritation of the nervous system produced by the tobacco.

The only effective treatment in any of this class of cases is that directed toward the relief of the underlying cause of the irritation of the nervous system. Local treatment of the eyes cannot be expected to produce much benefit.

Asthenopia Due to Reflex Causes

Sometimes we are able to trace the etiology of an asthenopia to an irritative or inflammatory condition in one or more of the abdominal organs, or to an abnormal condition in the nose. This may be termed a reflex asthenopia. It occurs in some cases of menstrual disorders, renal diseases, and other similar affections. In several obscure and obstinate cases that I have seen the diagnosis has been made and a cure effected by a thorough investigation of the gastrointestinal tract, followed by appropriate treatment of the condition found in that viscus. This form of asthenopia is quite apt to be associated with more or less imbalance of the ocular muscles, which may sometimes be simply another symptom of the disorder of the nervous system engendered by the organic trouble, so it is wise not to be in a hurry to ascribe the asthenopia to it. Careful observation is often necessary to make it certain whether gastric symptoms are caused by the eyes, or ocular symptoms by the stomach. Occasionally the heterophoria may be judged to be symptomatic from the unreliable action of the muscles, when the results of the tests vary a great deal on different days, or it may be on different hours of the same day, and the duction is subnormal.

In addition to the ocular troubles associated with diseases of the accessory sinuses asthenopia is quite often due to mechanical pressure in the nose, even when the patients present no nasal symptoms and are inclined to question the possibility that a fault in that situation can be the cause of their sufferings. The following case is typical of many. A seamstress, 31 years old, consulted me in January, 1893, because of headache and severe pain in the eyes, both of which were much aggravated by any attempt to sew or read. Her refraction was accurately corrected, she had orthophoria, and no morbid condition was present in either eye, except a chronic hyperæmia of the conjunctiva. The suggestion made by *Gruening* in 1886 that the cause of asthenopia might be in the nose led me to an examination of that organ, although she had no nasal symptoms. Pressure was found between the eburnated right middle turbinate and the septum. Portions of the turbinate were removed so as to relieve the pressure. Acute pain was felt in the right eye during the operation, but none in the nose. The headache and pain in the eye were relieved at once, but recurred several times when synechiæ were found to exist between the anterior end of the middle turbinate and the septum. The final division of these was followed by relief which lasted 19 years, until the spring of 1912, when a synechia was discovered to have formed in the same place from some unknown cause. Its division was again followed by complete relief. In this case, and in others like it, the place of contact was at such a point in the nose as to cause no noticeable obstruction to breathing, the pain was referred to the eye and brow, and nothing called the attention of the patient to the nose.

The clinical conditions are sometimes complex and confusing, as was the case in a patient who had compound hypermetropic astigmatism with the axis oblique in one eye alone, simple hypermetropia in the other, considerable hyperexophoria without correcting glasses, less exophoria and no hyperphoria with the glasses on if tested with the phorometer, or orthophoria by the cover test, slight bony contact between the right lower turbinate and the septum, and an adhesion between the septum and the right middle turbinate. Any of these conditions seemed competent to account for the headache and pain in the eyes from which the patient suffered, and the treatment had to be directed to more than one of them. Heterophoria was excluded as a probable cause by the marked improvement when the refractive error was corrected. Removal of the points of pressure in the nose was necessary, after the patient had worn accurately adjusted glasses for a while, and this, together with the correction of the refractive error, effected a cure which promised to be permanent two years later when the patient was last heard from.

Ziegler considers that the three most active nasal conditions in this class of cases are: 1, Pressure contact; 2, hyperæsthesia; 3, nasal obstruction. He says, "The first and most important causative agent is a condition which we may term pressure contact. The middle turbinate is usually the offending member, and is so often wedged in between the two vascular cushions of an engorged inferior turbinate and a sensitive septal puff that many mystifying reflex impulses are originated, the most pronounced of which are localized muscular twitchings, or choreiform movements of the face, head and neck, while intense frontal headache and eyeache are more frequent effects. The middle turbinate, however, is not always the offending member. I have seen an inferior turbinate cause similar disturbances which were promptly relieved by the removal of the spur. The pull of a contracting adhesion between the middle turbinate and the septum may also cause annoying reflexes.

"Another important etiological element is hyperæsthesia of certain areas in the upper air chambers of the nose. These sensitive points are most frequently located in an area covering the tubercle of the septum, which, when irritated, quickly becomes engorged with blood and thus makes pressure against the closely approximated middle turbinate. Irritation of this sensitive area is so provocative of distinctly localized eye symptoms that I have long since dubbed it 'the eyespot of the nose.'" The third element, nasal obstruction, is more likely to attract the attention of both the patient and the physician, but is of less importance as a cause of asthenopia as it acts indirectly, by increasing the sensitiveness of the whole system and rendering it more liable to reflex disturbances.

A diagnostic test on which I place considerable reliance is to press with a bit of wet cotton on an applicator upon the nasal mucous membrane of the septum at the point called "the eyespot of the nose," especially when it is in contact with the mucous membrane of the middle turbinate. If the pain caused in this way is of the same character as that for which relief is sought the patient recognizes it at once, and then the immediate removal of the pressure is indicated. The operation may be performed on either the turbinate, or the septum, according to circumstances. If the pain caused by such an application is purely local and of a different character from that of which the patient ordinarily complains it is doubtful if the asthenopia is due to the intranasal pressure in that particular case, and every other possible cause should be excluded before an operative intervention is undertaken, unless such intervention is indicated for other reasons.

Some of the More Recent Operations for Glaucoma

In spite of the fact that the presence of a cystoid cicatrix has long been considered a source of imminent danger to the eye, the opinion is widely prevalent at the present time that such a cicatrix is an essential feature in the success of any operation that may give permanent relief in glaucoma. Hence many operations have been, and are being devised to provide a permanent avenue of escape for the intraocular fluid, and at the same time to guard against the dangers found by experience to attend the presence of such a cicatrix, which have been described on page 502. All of these operations are modifications, in one way or another, of anterior sclerotomy, and the attempt is usually made to remove the dangers of irritation, rupture, and subsequent infection by covering the wound with a large flap of conjunctiva. They are all of such recent date that definite judgment cannot yet be passed as to their final results. We have yet to learn whether any or all of them are of permanent benefit, with regard to the course of the disease, and whether they do or do not render the eye more liable to succumb to some intercurrent trouble.

It is practically impossible to mention all of these operations, many of which differ only in some minor point, or have served as steps in the development of a technique, but three examples of methods which differ considerably may serve as competent illustrations.

In 1906 *Lagrange* introduced the operation of sclerectomy, which consists of an anterior sclerotomy with the formation of a large conjunctival flap, and the excision of a portion of the anterior lip of the scleral wound.

We instill eserine half an hour before the operation in order to contract the pupil and to draw the iris out of the way as much as possible. Then, after the eye has been cocainized, we introduce a *Graefe* knife 1 mm back from the limbus at a point about midway between the horizontal and vertical meridians of the cornea, pass it through the anterior chamber and make a counterpuncture on the opposite side, so as to make a section that will take in the upper fourth of the cornea. We hold the knife with its edge directed toward a point 2 mm behind the upper limbus, pass it by sawing movements through the angle formed by the iris and cornea, include as large a piece of sclera as possible in the anterior lip of the wound, and carry it backward for some distance, after it leaves this tissue, before cutting out, in order to form a large conjunctival flap. Next we lay this flap down over the cornea so as to bring the anterior lip of the scleral wound into view, which may need to be exposed still more by dissecting the conjunctiva from its surface. Then we excise this anterior lip with a pair of well curved, very sharp scissors held parallel to the wound and pressed downward. It is important that a sufficiently large piece of the entire thickness of the sclera be removed, so the scissors must be held firmly, with their edges so as to bite directly into the tissue, as otherwise only a part of the superficial layers may be removed, or the dense, resistant sclera may slip from between the blades. We must also be careful to see that the conjunctiva is not caught between the blades of the scissors, for if this happens a buttonhole will be made through which infection can enter the anterior chamber and no protection will be afforded by the large flap.

When the tension of the eye is greater than + 1, *Lagrange* recommends an iridectomy to be performed in the usual manner at this point in the operation, which is then called a sclerectoiridectomy.

The conjunctival flap is then replaced in its normal position, eserine is instilled, both eyes are bandaged, and the patient is put to bed for twenty four hours. The after treatment is the same as that for iridectomy.

Trephining of the sclera seems to have been suggested first by *Argyll-Robertson* in 1876, as a last resort in cases of glaucoma in which iridectomy had failed, but the operation has recently been popularized by Lieut.-Col. *R. H. Elliot*, I.M.S., who has greatly improved its technique and extended its application to all forms of the disease. At the present time it is very popular in the United States, and some enthusiastic advocates believe that it will supersede all other operations in the treatment of glaucoma. Other observers point to cases of quiet iritis and late infection as indicating that it is apt to be attended by serious dangers. Quiet iritis certainly seems to be a rather common complication, so much so that at least one advocate, *Peter*, advises that atropine be instilled immediately after the operation and for the three following days "to prevent posterior synechiæ from the quiet iritis which is sure to follow whether an iridectomy has been performed or not." Time alone can tell us the true value of this procedure and its limitations.

After anæsthetizing the eye we dissect up a large, triangular flap of conjunctiva with its base at the upper margin of the cornea. We elevate the base from the surface of the cornea, taking care not to buttonhole it, or else undermine the outer layers of corneal tissue for a distance sufficient to give room for the application of a trephine 2 to 2.5 mm in diameter at the corneoscleral junction. We cut or scrape away the subconjunctival tissue at this place until the sclera is perfectly bare, apply the instrument, taking care not to catch any fibers of the conjunctiva lest this be drawn into the wound, and cause it to rotate so that it will cut into and through the tissue. Some surgeons prefer a hand trephine, contending that they are able to control it by a better sense of touch, while others prefer one driven mechanically, as by a dental engine, because they think it inflicts less traumatism. As soon as the tissue has been cut through the aqueous wells up beside the instrument, and a peculiar sensation is communicated to the hand of the operator. The trephine and the button of tissue are then removed, and we observe the behavior of the iris. If it occupies its normal position we have only to replace the conjunctival flap and the operation is completed, but if it bulges into the opening, as it is quite apt to do, we buttonhole it with scissors. If it still refuses to return to its normal position an iridectomy, either partial or complete, is necessary. A partial iridectomy includes only the peripheral portion of the iris. The conjunctival flap is then smoothed back in place, where it may be secured by one or more sutures if the surgeon thinks best. Eserine may be used later if the iris shows a tendency to prolapse.

Modifications of the technique are endless. *Fergus* adds a cyclodialysis; *Johnson* splits the sclera and cornea with a keratome, punches out a quadrilateral piece at the corneoscleral junction, and abandons the protective conjunctival flap; *Fox* forms the conjunctival flap like *van Lint*.

In the T-shaped sclerotomy, advocated by *van Lint* in 1913, after an experience of about a year, the large conjunctival flap is dissected away from the cornea and later brought up over the field of operation, while the wound is caused to gape without the removal of any of the scleral or corneal tissue.

The first step in the operation is to dissect away the conjunctiva from all around the upper half of the cornea, so as to expose the sclera for a distance of at least 1 cm; the second is to insert a suture on each side of the cornea in such a way that when they are tightened the conjunctival flap will be drawn over the upper fourth of the cornea. The lower end of each suture is passed beneath the conjunctiva at the extremity of the dissection to emerge 2 mm lower down; the upper end is passed through the margin of the detached conjunctival flap about 10 mm from the place where the lower end was introduced. These sutures are left loose, the flap drawn back from the sclera, and the horizontal portion of the T-shaped incision is made with a keratome 2 to 2.5 mm from the limbus, where the knife will enter the eye behind the iris. The iris is usually punctured by the knife, thus producing an iridodialysis. *Van Lint* advises that an iridectomy be performed in acute or subacute glaucoma, or a partial, peripheral, one in some of the chronic forms. As the iris is often in contact with the cornea, and as the wound is posterior to the iris, it is advisable to use a forceps provided with a tooth on its concave side. One blade of a pair of slender, straight scissors is then introduced into the scleral wound and a vertical cut made from its middle to a point in the cornea 1 to 1.5 mm from the limbus. He says that at the moment this is done the lips of the wound separate with force, as if some elastic fibres existed at that spot, and a relaxation of the eye is produced. The conjunctival flap is then drawn down so as to cover the entire wound and secured in place by tying the sutures on each side of the cornea. The sutures may be removed on the fifth day; then the conjunctival flap will gradually draw back to its normal situation. The eye is bandaged for a few days and a myotic instilled for some weeks.

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